BRAIN:

A JOURNAL OF NEUROLOGY.

VOL. IV.

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A JOURNAL OF NEUROLOGY.

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VOL. IV.

APRIL 1881 TO JANUARY 1882.

London:

MACMILLAN AND CO.

1882.

LONDON:
PRINTED BY WILLIAM CLOWES AND SONS, LIMITED,
STAMFORD STREET AND CHARING CROSS.

CONTENTS.

ORIGINAL ARTICLES:—

CRITICAL DIGESTS AND NOTICES OF BOOKS:—

CLINICAL CASES:—

ABSTRACTS OF BRITISH AND FOREIGN JOURNALS:—

LIST OF CONTRIBUTORS.

ALTHAUS, JULIUS, M.D.
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GORHAM, JOHN, M.R.C.S.
HOBSON, J. M., M.D.
HUGHLINGS-JACKSON, J., M.D.
IRELAND, W. W., M.D.
JAMES, ALEXANDER, M.D.
KRUEG, DR. JULIUS.
LEWIS, BEVAN, L.R.C.P.
MACLEOD, M. D., M.D.
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RATTON, J. J. L., M.D.
RIVINGTON, WALTER, F.R.C.S.
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URQUHART, A. R., M.D.
WALLER, A., M.D.
WARNER, FRANCIS, M.D.
YANDELL, DAVID W., M.D.

LIST OF ILLUSTRATIONS.

BRAIN.

APRIL, 1881.

Original Articles.

THE LATE LORD CHIEF JUSTICE OF ENGLAND ON LUNACY.

BY JOHN CHARLES BUCKNILL, M.D., F.R.S.

ONE has recently passed from among us in the full ripeness of years and of honours, who has left a deep impression upon our memories, of the force and versatility of his character, and of the decided and sometimes original opinions which he had formed and maintained upon many of the subjects which fell within the scope of his wide influence. For in many of the most interesting dramas which for many years past have been presented on the political or the forensic stage Sir Alexander Cockburn played one of the leading parts, and it especially fell to his lot to be engaged in contentions whose issues, if they have not greatly promoted the science of psychology, have at least gone far to shape and determine the practical question of the relations of the insane with society and its laws, both with regard to their exemptions and their disabilities.

Among the former, the most remarkable and memorable was his defence of McNaughten for shooting Mr. Drummond, on the plea that he was not guilty on the ground of insanity. This *cause célèbre* has been worn threadbare by discussion, but in looking over Cockburn's very able advocacy we cannot fail to notice his generous recognition of the great value of medical knowledge and experience in determining such difficult questions as the one before the Court; a fixed state of feeling and belief which, to the last days of his long life, he rarely

failed to express whenever the opportunity offered, in observations which form a notable contrast to such remarks disparaging to medical science as are even now sometimes heard in Courts; remarks which tend to show, if they mean anything, that great lawyers sometimes have not the wit, or will not take the pains, to distinguish real science from false pretensions to it. In McNaughten's defence Mr. Cockburn said: "It was now placed beyond doubt that madness was a disease of the body, the result of morbid organization, and that its nature was to be precisely and accurately ascertained by those only who had made this disease and its pathology the object of long reflection and diligent investigation. The discoveries of modern science had thrown much light upon this subject, and many of the positions laid down by Lord Hale and the other authorities of former times were left liable to very great objection and doubt."[1]

In addition to the stress which he laid upon the value of scientific knowledge, a remarkable feature in his defence of McNaughten is apparent in his early recognition of the element of loss of self-control. He undertook to prove to the jury that the prisoner "was the creature of delusion and uncontrollable impulse, which took away from him the character of a responsible being." This term "uncontrollable impulse" has since been very greatly misused and abused, inasmuch as it has been attributed to offenders who had no other characteristics of insanity; and, as we shall see, Cockburn subsequently adopted the better term of "absence of the power of self-control." While we must repudiate the overstrained doctrine that uncontrollable impulse is in itself and by itself, a form of insanity, we must recognise the fact that the quality is essential to the commission of an offence by a madman. Whether we say that the man is impelled to do a thing by his madness, or whether we say he did that which his mad thoughts led him to do in "the absence of self-control," or as another great judge has put it, that he did that in his madness which he could not help doing, we have the same thought with some little variety of expression. If we could know that a person having a delusion could so have controlled himself as not to

[1] The 'Times,' March 6th, 1843.

have indulged or acted upon it, our view of his responsibility would be very different to that which we must take in regard to "a creature of delusion" deprived of all self-control. The practical difficulty, of course, is that of deciding when, in consequence of disease of the organism, a man is deprived of self-control or has an uncontrollable impulse, a difficulty certainly not less than that of deciding when a person of unsound mind does or does not retain the knowledge of right and wrong with regard to his actions; but why the difficulty should be thought by our legal authorities to be insurmountable in the one case but not in the other, it is not easy to understand. We must briefly refer to Cockburn's opinion on this important point, and also to that of his compeers who were not able to concur in it.

Thirty-one years after the McNaughten trial, the counsel for the defence, now become Lord Chief Justice of England, repeated the opinions maintained in that trial, in all the maturity of his judgment and with all the weight of his authority, in his Memorandum to the Chairman of the Select Committee of the House of Commons on the Homicide Law Amendment Bill. In this important document he says:

"As the law, as expounded by the Judges in the House of Lords, now stands, it is only when mental disease produces incapacity to distinguish between right and wrong, that immunity from the penal consequences of crime is admitted. The present Bill introduces a new element, the absence of the power of self-control. I concur most cordially in the proposed alteration of the law, having been always strongly of opinion that, as the pathology of insanity abundantly establishes, there are forms of mental disease in which, though the patient is aware he is about to do wrong, the will becomes overpowered by the force of irresistible impulse, the power of self-control, when destroyed or suspended by mental disease, becomes (I think) an essential element of responsibility."

There would seem to be a clerical or printer's error in this last sentence, the context clearly showing that the writer intended to say that the suspension of the power of self-control was an element of irresponsibility. And no doubt it is; the difficult question remaining to be solved as to what are the forms

or degrees of mental disease in which the power of self-control is suspended or destroyed. For instance, in general mania, with or without delusion, the power of self-control is thus suspended; but is this the case also in what is called homicidal mania, as it is described without the group of symptoms which usually accompany diseases affecting the mind? It is much to be wished that the Chief Justice had expressed his opinion more fully and clearly on this most important point. In a subsequent passage, however, towards the end of his memorandum he does connect other symptoms of insanity, that is to say delusions, with the loss of self-control in such a manner that it must be inferred that in the passage already quoted, he did not intend to endorse the dangerous and illogical theory of so-called moral insanity. In this later paragraph he says:

"I have already expressed my concurrence in the proposed provisions as to the effect of insanity, except so far as the proposed legislation is partial, as limited to the case of homicide. But there is one general provision on this subject to which I must strenuously object; it is that, 'if a person is proved to have been labouring under any insane delusion at the time when he committed homicide, it shall be presumed, unless the contrary appears to be proved, that he did not possess the degree of knowledge or self-control hereinbefore specified.' The pathology of insanity shows that the mind may be subject to delusions, which do not in any degree affect the moral sense or the will as regards the power of self-control. The mere existence of mental delusion ought not to affect the decision as to the power of self-control, unless the nature of the delusion be such as would legitimately lead to the inference that the power of self-control was wanting. The question is one which should be decided by all the circumstances, independently of any presumption one way or the other."

It will be seen, when we come to comment upon this Chief's most important judgment in a civil cause, how consistent he was in his views of what we may perhaps be permitted to call the autonomy of delusion. It may be that his views were extreme in this regard both in criminal and civil law. Correct in his fact that insane delusion may exist without the loss of self-control, he scarcely seems to have given weight enough

to the importance of delusion as evidence of insanity more or less general; and we venture to assert that where a man who has committed a homicide is proved to have been labouring under any insane delusion at the time, the inference will inevitably present itself that with the loss of the power of reasoning the man has also lost the power of self-control. Whether in such cases the legal burden of proof should be shifted, as proposed by the framers of the Bill of 1874, is quite another and an important question. It is possible that an insane delusion may not affect a particular action; but seeing that an insane delusion indicates grave mental disease, and that it is impossible to say how far its influence extends and where it stops, there must always exist great doubt as to any action being free from its influence. It must at least be extremely difficult to prove that any deliberately wrong, and especially any penal action, was not done under the influence of the delusion. Therefore this shifting of the burden of proof proposed would be tantamount to making the proof of delusion exonerate the crime. One cannot get on without presumptions, and many of them, in deciding such difficult and often recondite questions as those depending upon the influence of diseased mind upon conduct; and if the presumption of the wide-extending influence of insane delusion be admitted, it would surely be more wise in conception and simple in practice to extend its exonerating influence than to change the burden of proof in the manner proposed. The recent Royal Commission of four pre-eminent lawyers employed to draw up the Criminal Code decided against the section of the Criminal Code Indictable Offences Bill of 1878 which recognised as an excuse the existence of an [insane] impulse to commit a crime, on the ground that the test proposed [namely the supposed inefficiency of the greatest and most immediate punishment] for distinguishing between such a state of [insane] mind and a criminal motive would not be practicable or safe. In the draft Criminal Code drawn up by the Royal Commissioners, Blackburn, Barry, Lush, and Stephen, all reference to loss of control is omitted, and the existing law with regard to the pertinence of delusion to the crime is left as it was; only it is proposed newly to provide that any insane

delusions, including, of course, such as are not pertinent to the crime, may be given as evidence that the offender was in such a condition of mind as to enable him to be acquitted on the ground of insanity; that is to say, that he was labouring from natural imbecility or disease affecting the mind to such an extent as to be incapable of appreciating the nature and quality of the act, or that the act was wrong. Without doubt it is extremely difficult to formulate such a legal doctrine as that of the loss of self-control as an excuse for crime, so that it shall not be dangerous to society; but seeing that the knowledge of right and wrong can have no effect on the commission or omission of crime, unless the agent has the power to control himself so as to commit or omit the action which constitutes a crime, and seeing that madness does enormously affect this power, we cannot but think that the late Chief Justice came far nearer to a right and satisfactory solution of this difficult problem in the objections and suggestions of his memorandum than the Royal Commissioners.

In the letter which in June 1879 the Chief Justice addressed to the Attorney-General on the Criminal Code Bill, he has criticised the law as it exists, and its proposed alterations, in a manner which must increase the difficulties which judges cannot fail to experience in charging juries whenever the plea of "not guilty, on the ground of insanity," is put forward. His own opinions, however, are not so clearly expressed as to leave it without doubt what he really meant, especially on the extremely important question of homicidal insanity, or of moral insanity generally. In a recent work on insanity ('The Factors of Unsound Mind,' by Dr. Guy), his authority is quoted from this letter in support of the theory of "instinctive or impulsive homicidal monomania," and his language is certainly open to the interpretation which this author puts upon it. Thus he says:—"Among the functions of the human mind liable to be perverted by disease is, as all scientific writers on insanity are agreed, the human will, which sometimes becomes the slave of maniacal impulses, which it is unable to resist. Among the different forms of madness by which the will is liable to be thus affected, is that which is known by the term of homicidal mania, or, when it impels a person to self-destruc-

tion, suicidal mania. That the will is liable to be thus maniacally affected, and so to be swayed by impulses which it is unable to resist, is a point on which writers on mental pathology are agreed." "The question whether, under the influence of mental disease, the human will may become subject to impulses which it is unable to resist, and upon which even the fear of death will not operate as a restraint, is not one for lawyers to dispose of dogmatically, as they too often do, but one which, as a question of pathological science, it is for men conversant with that science to decide." "The question whether *mania, accompanied by insane impulse*, might afford a defence, was not submitted to the Judges [in the McNaughten case], or involved in their answers."

Now it may be argued that these passages, with the context, affirm the doctrine of insanity of the will, or at least that they affirm the existence of homicidal and suicidal monomania, as dependent upon insane impulse alone. But it is to be noted that the Chief Justice invariably speaks of these affections as *maniacal;* thus, "the will becomes the slave of maniacal impulses," and "the will is liable to be maniacally affected," and "the question is whether mania accompanied by insane impulses," &c. Nowhere does he say that the will swayed by impulses unaccompanied by the symptoms of mania, is a form of insanity, or might afford a defence. But those writers who maintain the existence of homicidal and suicidal, or of the stealing, or ravishing, or house-burning forms of so-called moral insanities, do describe them as unaccompanied by the general symptoms of mania, mental and physical. Of such forms of insanity a considerable number of "writers on mental pathology" utterly deny the existence, although all such writers will, and do, undoubtedly admit that such conduct accompanies real mania. Seeing therefore that the Chief Justice asserts that his statement of facts is a "point on which writers on mental pathology are agreed," it may fairly be argued that by the impulsive affections of the will which he describes, he meant the headstrong conduct of the real maniac, which no one denies, and therefore that he was not a convert to the new doctrine of moral insanity, which never yet has withstood the trial of experience or the test of argument.

That will-o'-the-wisp, the human will, has misled metaphysicians and moralists often enough, to make us heartily desire that at least grave and reverend Judges should be blind to its glamour. And by omitting such speculations as that "the human will becomes the slave of maniacal impulses," "which it is unable to resist," &c., we may gather that the Chief Justice intended to express his conviction that a man having no delusions might be in a maniacal condition, accompanied by insane impulses to the commission of offences against the law, and that the proof of such a condition ought to be a valid defence. This "is a point on which writers on pathology are agreed," it being understood that the maniacal condition connotes a well-known group of symptoms called mania, in persons who are properly said to be "maniacally affected."

This is "the question which was not submitted to the Judges, nor involved in their answer," and upon which the late Chief Justice expressed his opinion in such metaphysical arguments, as leave his meaning needlessly obscured, but which lead to the conclusion, as entertained by him, that a maniac, even without delusion and with knowledge of right and wrong, is nevertheless irresponsible.

This great Judge has left the mark of original thought and decision still more deeply impressed upon the civil than on the criminal side of lunacy in the Courts. As an advocate, one of his most brilliant efforts was made in his successful opposition to his distinguished opponent Sir Frederick Thesiger, in the cause of Sefton *v.* Hopwood, tried before Mr. Justice Cresswell at the Lancashire Assizes in 1855. The Earl of Sefton propounded the will on behalf of the younger children of the testator, his father-in-law, and Mr. Cockburn opposed it on behalf of the elder son, who had been disinherited. Cockburn admitted that in this case there was no evidence of delusion, which in McNaughten's case he had followed Erskine in declaring to be the essential feature of lunacy. But he reasoned, and he proved that there was abundant evidence of loss of memory and of confusion of thought extending, as by his masterful eloquence he persuaded the jury to believe, to complete testamentary incapacity. In this trial, as in McNaughten's, he gave the full meed of commenda-

tion to the medical evidence, and justly so, for the medical attendant of the great county family whose dearest interests were involved had behaved with resolution under trying circumstances in refusing to witness the will, and had given his evidence with a judgment and temper which could not fail to influence the jury towards his honest and well-considered opinion.

In the following year Sir Alexander Cockburn led in maintaining the validity of the will in the great Swynfen case. But the arguments or the statements of opinion expressed by advocates have perhaps their greatest permanent value as preparations or training of the judicial mind, upon which the determination of the law eventually depends. It may be interesting and not unprofitable in such a man to trace the arguments of the barrister in the judgments of the Judge, but the more important duty of endeavouring to estimate the full nature of the more important of these judgments must check diffuseness on such matter as does not bear the stamp of authority.

In Banks *v.* Goodfellow, the matured opinions of the Chief Justice are expressed on insanity in its relation to testamentary capacity, and this judgment is remarkable on account of its forming one of the rare instances in which all accepted precedents of legal authority have been reversed. The "doctrine" of the Courts had been that "any degree of mental unsoundness, however slight and however unconnected with the testamentary disposition in question, must be held fatal to the testamentary capacity of a testator," and this doctrine, Chief Justice Cockburn said, had been very emphatically declared by Lord Chancellor Brougham, presiding over the Judicial Committee of the Privy Council in Waring *v.* Waring. The head-note to this remarkable case, 6 Moore, P. C. Rep., thus explains the purport of the judgment:—

"This exposition of the doctrine of monomania and partial insanity, as applied to wills, was that if the mind is unsound on one subject, providing that unsoundness is at all times existing on that subject, it is erroneous to suppose such a mind is really sound on other subjects; it is only sound in appearance, for if the subject of the delusions be presented to it,

the unsoundness would be manifested by such a person believing in the suggestions of fancy as if they were realities, any act therefore done by such a person, however apparently rational that act may be, is void, as it is the act of a morbid or unsound mind."

"This doctrine," said Cockburn, "was, perhaps, more fully expressed by Lord Penzance in Smith *v.* Tebbitt." Thus: "A person who is affected by monomania, although sensible and prudent on subjects and occasions other than those upon which his infirmity is commonly displayed, is not in law capable of making a will. . . . For I conceive the decided cases to have established this proposition, that if disease be once shown to exist in the mind of a testator, it matters not that the disease be discoverable only when the mind is addressed to a certain subject to the exclusion of all others; the testator must be pronounced incapable. Further, the same result follows, though the particular subject upon which the disease is manifested have no connection whatever with the testamentary disposition before the Court."

This legal doctrine was abolished as wrong, and a contrary rule substituted in its stead by the judgment of Chief Justice Cockburn, presiding in Banco of Queen's Bench, Justices Blackburn, Mellor, and Hannen concurring therein, upon the motion for a new trial in Banks *v.* Goodfellow, July 6, 1870. The testator John Banks was admitted to have been of unsound mind; he had been confined in an asylum and remained subject to certain fixed delusions. He had conceived a violent aversion to a man long since dead, whom he believed still to pursue and molest him, and the mention of this man's name threw him into violent excitement. He frequently believed that he was pursued and molested by devils or spirits, whom he believed to be visibly present. There was conflicting evidence as to his general insanity, but "from September [the will being made on December 2 following] 1863, he had a succession of epileptic fits, and a blister was applied to his head, and the medical man who attended him throughout this period deposed that his mental power, such as it was, suffered from these fits, and that he considered the testator insane and incapable of transacting business during the whole time." On

the other hand, it appeared that he managed his limited affairs and was careful of his money. The Westmoreland jury found for the validity of the will, which in-so-far as it bequeathed his property to his niece who lived with him was a natural disposition of his property; but, inasmuch as his niece was his heir-at-law and would have inherited without the will, it was a vain and unnecessary one. The niece died shortly after the testator, and her heir, being no kin to the testator, claimed under the will, thus disinheriting the testator's nephew, the plaintiff, who strove to cancel the will on the ground of the incapacity of the testator.

From this statement, somewhat abbreviated from that made by the Chief Justice himself, it will appear to any practical psychologist, that in order to reduce the issue to a question of principle, extremely large assumptions were made in the judgment, in that it assumed the absence of general insanity, and also that "the delusions must be taken neither to have had any influence on the provisions of the will, nor to have been capable of having any." Thus trimmed to the most convenient condition for argument, of course the question became "whether a delusion, thus wholly innocuous in its results as regards the disposition of the will, is to be held to have had the effect of destroying the capacity to make one." The simple dogma of the older jurists that an insane person was incapable of making a testament "*quia mente caret*" is obviously unsatisfactory in the opinion of the Chief Justice, "when the fact becomes recognised that a man may labour under *harmless delusions which leave the other faculties of his mind unaffected*, and leave him free to make a disposition of his property uninfluenced by their existence." The judgment, therefore, with regard to direction was that "a jury should be told in such a case that *the existence of a delusion compatible with the retention of the powers and faculties of the mind will not be sufficient to overthrow the will, unless it were such as was calculated* to influence the testator in making it;" and, looking at the evidence by the light of this doctrine, the Court refused a new trial, on the ground that "in our judgment the only proper or possible result must be a second verdict establishing the will."

In order that we may the better appreciate Chief Justice Cockburn's argument and the judgment founded upon it, the judgment in Waring *v.* Waring delivered by Lord Chancellor Brougham, with the concurrence of Lords Langdale, Lushington and Pemberton Leigh, of the Judicial Committee of the Privy Council, ought to be considered; seeing that it is the faultiness of this opposite view, of which this decision is the most remarkable and authoritative expression which is believed to have justified its reversal. Lord Brougham said that, in speaking of disease or decay affecting the mental faculties being more or less general, or of affecting more or fewer of these faculties, "we must always keep in view that which the inaccuracy of ordinary language induces us to forget, that *the mind is one and indivisible,* and that when we speak of its different powers or faculties, as memory, imagination, consciousness, we speak metaphorically, likening the mind to the body, as if it had members or compartments, whereas, in all accuracy of speech, we mean to speak of the mind acting variously, that is remembering, fancying, reflecting, the same mind in all these operations being the agent."

A man's mind being one, Lord Brougham says that cases of monomania are incorrectly called partial insanity. "We are wrong in speaking of partial unsoundness, we are less incorrect in speaking of occasional unsoundness; we should say that the unsoundness always exists, but it requires a reference to the peculiar topic, else it lurks and appears not. But this malady is there, and the mind is one and the same; it is really diseased while apparently sound, and really its acts, whatever appearances they may put on, are only the acts of a morbid and unsound mind." "We never can rely on such acts, however rational in appearance, because we have no security that the lurking delusion, the real unsoundness, does not mingle itself with or occasion the act." This latter point, not in any way dependent upon metaphysical opinions or doctrines, will be most readily admitted by those who have the largest experience of the insane. One can never tell when their insanity will not crop out; so that their testamentary and other acts, which may appear to be quite reasonable, may have an insane motive, and indeed are rarely altogether

free from some admixture of such motive; and Brougham's observation is perfectly justified that "it is hardly possible that any will can be so framed as to rebut all presumptions of insanity arising from proved facts," for all the conduct of the insane is tainted with at least suspicion of insane motive; nay, the presumption is and always must be that there is some degree of insane motive in all their conduct, or, in other words, that the motive of an insane man never is quite what it would have been had he been of sound mind. Not that the motive is altogether perverted and different, but that, looking to the full composition of motive in memory, fancy, feeling and desire, and all the other faculties which go to influence conduct, the motive of the most typical monomaniac, whether it be determined to call him insane as to parts of his mind, or insane as to occasions of thought, or insane as to a limited range of things or affairs, cannot be relied upon as a sane motive, even if the act be apparently that of a sane man.

On this point Lord Brougham's opinion is inexpugnable, but not so in regard to his objection to the term "partial insanity," on the ground that there is no such thing, seeing that the mind being one, all insanity must be held to affect the whole mind. It is much more true that all insanity is partial insanity, and that there is no such thing as insanity which is not partial. As the existence of bodily disease implies the continuance of life, so the existence of insanity implies the continuance of mind. Therefore coma, and perhaps the extreme of amentia, can scarcely be called insanity, which is perversion or defect of mental activity, not its abrogation. But partial insanity, as it was understood and objected to by Brougham, was not this kind of partiality. It was the partiality with which we speak of local disease of the body in contradistinction to constitutional or general disease, and his objection to it on the ground that mind is one and indivisible, and therefore that its affections cannot be limited to a part or parts, although an argument of the metaphysical kind, is still one which can scarcely be avoided in thorough discussion of such a question. Indeed it would seem that we are somewhat less metaphysical, and more in accordance with the common sense and opinion of

mankind, when we say with Brougham that mind is one thing, and that this habit of speaking of its various activities as if they were distinct things is but a metaphorical mode of expression due to the imperfection of language, than if we were to adopt Cockburn's view of the independence of the faculties.

In the Banks *v.* Goodfellow judgment, Chief Justice Cockburn "did not think it necessary to consider the position assumed in Waring *v.* Waring, that the mind is one and indivisible, or to discuss the subject as matter of metaphysical or psychological inquiry. It is not given to man to fathom the mystery of the human intelligence, or to ascertain the constitution of our sentient and intellectual being." And yet he immediately proceeded to discuss the question in a metaphysical or rather psychological manner as follows:—"Whatever the essence of it may be, every one must be conscious that the faculties and functions of the mind are various and distinct, as are the powers and functions of our physical organisation. The senses, the instincts, the affections, the passions, the moral qualities, the will, perception, thought, reason, imagination, memory, are so many distinct faculties or functions of the mind. The pathology of insanity and the experience of its various forms teach us that while, on the one hand, all the faculties, moral and intellectual, may be involved in one common ruin, as in the case of the raving maniac, in other instances one or more only of these faculties or functions may be disordered, while the rest are left unimpaired and undisturbed; that while the mind may be overpowered by delusions which utterly demoralize it and unfit it for the perception of the true nature of surrounding things, or for the discharge of the common obligations of life, there often are, on the other hand, delusions which, though the offspring of mental disease, and so far constituting insanity, yet leave the individual in all other respects rational, and capable of transacting the ordinary affairs and fulfilling the duties and obligations incidental to the various relations of life. No doubt when delusions exist which have no foundation in reality and spring only from a diseased and morbid condition of the mind, to that extent the mind must necessarily be taken to be unsound; just as the body, if any of its parts or functions is affected by local disease, may be said to be unsound, though

all its other members may be healthy and their powers or functions unimpaired. But the question still remains whether such partial unsoundness of the mind, if it leaves the affections, the moral sense, and the general power of the understanding unaffected, and is wholly unconnected with the testamentary disposition, should have the effect of taking away the testamentary capacity."

Further on he declares that the fact must be recognised that "a man may labour under harmless delusions, which leave the other faculties of his mind unaffected, and leave him free to make a disposition of his property, uninfluenced by their existence." Surely the above quotation will justify us, not only in declaring that it is impossible to discuss such subjects except as matters of psychological inquiry, but that the Chief Justice did so discuss them, and did not discuss them well, in consequence of his abortive attempt to assimilate mind more closely than in the nature of things will bear to be done to the functions and even to the parts of the body. There is no real resemblance or community of nature between a delusion and a part of the body affected by local disease; neither is it true that a delusion is a faculty of the mind, as Cockburn appears to think when he speaks of delusions and other faculties of the mind. The argument which compares a delusion to an injured hand, or an eye which a man can pluck out and cast from him, needs no refuting; but to consider a delusion one of the mental faculties is a serious error, inasmuch as it seems to vitiate the whole of an argument in many respects acute, able, and learned. It is, in his own forcible language, "a position obviously unsatisfactory when the fact becomes recognised" that a delusion is a state of mind in which all the mental faculties or activities are affected. Sense, perception, judgment, feeling, and whatever else goes to constitute mind are, and must be, each and all affected in such a fundamental perversion of the reason. In the concrete instance discussed, John Banks, the testator, who had conceived a violent aversion to Featherstone Alexander, and, notwithstanding the death of the latter, believed that he still pursued and molested him, and the mention of whose name threw him into violent excitement, and who believed that he was pursued and molested by devils who were visibly present to

him, must have had perversion of the sense and perception, constituting hallucination; must have had the faculty of comparison perverted, or he would have recognised the false sense, as Nicholai and others have done; must have had his feeling perverted, or he would not have been thrown into violent excitement by the chimera of persecution by a man long since dead, but whose death he could not remember. The mental activities implicated in these beliefs were as little partial, as regards the field of mind which they implicated, as the mental activities of the Chief Justice himself, in the judgment which he delivered upon them, or as those of the Judges whose opposite judgments he superseded. It is a marvellous thing that a man can be stark staring mad upon one train of ideas, and that you can start the manifestation of his madness by a word, while upon all other topics and trains of thought he is comparatively rational and seemingly of sound mind. But it is of the first importance to discussions on the influence of delusions, either on responsibility or on testamentary capacity, to be assured that not part of the mind but the whole of a man's mind, whatever we may think of its unity or its composition, is affected by such a delusion, which absorbs the man while he is excited by it; not one of his faculties to the exclusion of others, but the whole of his faculties on the particular subject or subjects respecting which he entertains insane delusions. We cannot but think that Chief Justice Cockburn misapprehended and therefore misstated the argument of Lord Brougham in Waring *v.* Waring, and that of Lord Penzance in Smith *v.* Tebbitt, since he says that the doctrine for the first time laid down by them "may be shortly stated thus. To constitute testamentary capacity, soundness of mind is indispensably necessary. But the mind, though it has various faculties, is one and indivisible. If it is disordered in any one of those faculties, if it labours under any delusion arising from any such disorder, though its other faculties and functions may remain undisturbed, it cannot be said to be sound. Such a mind is unsound, and testamentary incapacity is the necessary consequence." The doctrine referred to really appears to have been so much opposite of this, that it would be far more correct to state it thus. The mind being one and

indivisible cannot be disordered in any one of its faculties; and mental disease implies disorder of all. Insane delusion affects all the mental faculties, although such affection may be latent when the subject of delusion is not before the mind. Such a mind is unsound, and testamentary incapacity is the necessary consequence. It was not argued by Lords Brougham and Penzance that insane delusions on a limited subject of belief and feeling must necessarily affect the motives of a man making a will, but it was argued that insane delusions would cast such a doubt on those motives, and would moreover throw such a taint of suspicion upon the soundness of the understanding of the person so affected as to amount to testamentary incapacity. Chief Justice Cockburn, on the other hand, assumed as the foundation of his argument that insane delusions might exist, and in the case under consideration did exist, "delusions which had, in point of fact, no influence whatever on the testamentary disposition in question," "delusions which had not, nor were calculated to have any influence on him in the disposal of his property;" "and the question is whether a delusion thus innocuous in its results as regards the disposition of the will, is to be held to have the effect of destroying the capacity to make one."

If this line of argument is not begging the question it is very like that common fallacy, for if it be admitted that the delusion must be taken neither to have had any influence on the provisions of the will, nor to have been capable of having any, *cadit quæstio.* But if such admission be not made, as "in this case we are dealing with," it certainly ought not to have been made, it is the argument which fails. "In this case we are dealing with," the delusions, as described by the Chief Justice, might have influenced the testator's disposition of his property in an infinite variety of ways. Take one of the most probable, namely that he was so preoccupied by his delusions that he did not recollect the existence or the claims of the nephew, who was not present, but only those of the niece, who was present, for it is a frequent effect of delusion to exclude reflection upon subjects not within its range. And again, in considering "this case we are dealing with," what is more probable, nay, almost certain, than that the only relative

living with a person labouring under such delusions of persecution by a dead man, and by devils and evil spirits, would become intimately related with those delusions, although they did not directly include or refer to herself? Would she not have had to comfort him under his fanciful terrors, to assume the *rôle* of protector against his persecutors, and would such a position have no effect upon the dispositions of the lunatic? Clearly this reasonable probability was quite opposite to that which the Chief Justice so resolutely assumed as the certainty, for he did not put it as a probability but as a certainty, that the delusions could have had no influence whatever on the testamentary disposition.

We venture therefore to think that the argument of Chief Justice Cockburn did not quite meet, and that certainly it did not entirely answer, the argument of Lords Brougham and Penzance with regard to the influence of delusion on testamentary capacity. At the same time we cannot fully concur in the conclusions of Lords Brougham and Penzance. They may follow the psychological premises, but they are too formal and definite to adapt themselves to the infinite varieties of Nature, even as she is seen in the aberrations of the human mind. Perhaps less injustice would be done under their rule of exclusion than by Chief Justice Cockburn's rule of admitting the wills of lunatics with delusion, for without doubt a great and increasing amount of injustice is done every year by the probate of the wills of lunatics which ought never to have been made. It is a great social privilege, that of bequeathing one's property to whomsoever or for whatsoever one thinks fit, possessed in its unfettered condition by the English race almost alone among the nations, and no one can peruse the eloquent remarks which the Chief Justice made in this judgment upon the moral responsibility of exercising this privilege, or rather of discharging this duty aright, without an increased feeling that its abuse ought to be strictly guarded. At the present time the legal doctrine as to testamentary capacity and incapacity does not appear to be in a satisfactory state; seeing that the old doctrine, accepted for a long period and reaffirmed perhaps upon insufficient argument, has been upset, and an opposite doctrine substituted upon argument far from satisfying and

conclusive. It may at least be objected to all the argument on both sides that far too much stress has been laid upon delusion, as if it were the main element of insanity, and as if it were always very much the same kind of thing in nature and degree, only applied to different kinds of subjects. With all of those great Judges a delusion is a very definite and positive state of belief. It is wrong belief; but it is so strong that a man who entertains it is sure to act upon it. But, we will not say with Cockburn, "those who are most conversant with the pathology of mental disease," but use the better term with which he follows up that too flattering phrase, "those who have most experience of insanity in its various forms," well know that insane beliefs are like sane beliefs, in all their infinite variety of strength or weakness, clearness or obscurity, persistency or changeableness, activity or latency, power or incapacity to influence action; and that they have, in almost as great a variety as sane beliefs, connection with or independence of an infinite variety of passions or emotions, and of intellectual conditions other than beliefs. A man may possibly have a delusion which would seem extremely likely to influence his will, and yet from some quality in the nature of his belief in his delusion, it shall not influence his will or seriously affect his conduct in life. On the other hand, another man may well have a delusion which Chief Justice Cockburn would have been too ready to say "was wholly innocuous as regards the disposition of his will," and yet, through one of those cross-cuts of perversity so rife in the insane mind, the will shall be nothing but the expression of the delusion. These facts, which will be in agreement with the experience of all competent observers, point to the conclusion that testamentary incapacity ought to be decided upon, not by the existence of delusions or their supposed interference or non-interference with the reasonings or feelings of the testator, but upon all the circumstances of each individual case, which is the identical doctrine advocated by Cockburn himself with regard to the influence of delusion in determining irresponsibility for criminal acts.

But in this judgment the Chief Justice went beyond this reasonable doctrine, when he so far depreciated the importance of delusion as to declare that the opinion must be looked upon as

merely speculative, and unsupported by proof, which assumed the probability that where insane delusion has shown itself, a greater degree of mental unsoundness exists than has actually become manifest. It surely is not a matter of merely speculative opinion, but one rather of pretty constant experience, that where fixed delusions exist the mind is profoundly affected. In this very case "the mere mention of Featherstone Alexander's name [the dead man who still pursued and molested him] was sufficient to throw him into a state of violent excitement." "But as the delusion was not manifested at the time of making the will, it is a question whether the delusion was not latent in the mind of the testator." Just so. There was a greater degree of mental unsoundness than was actually manifest. There was delusion and a morbid state of temper and feeling which a word could at any hour explode into violent excitement.

But it must be admitted that some delusions do not indicate any greater degree of mental unsoundness than that which is always manifest, and that there are some delusions which may be predicated as unlikely, though not incapable, of having any effect upon a will. The varying delusions of hypochondriasis are quite different from the fixed delusions of persecutions such as were entertained by Thomas Banks; and while these would not necessarily indicate any mental unsoundness beyond the unfounded and extravagant opinions they represent, the others would necessarily indicate profound mental lesions which would be almost certain to manifest themselves in aberrations and defects of the understanding, and in perversions of the affections on other subjects than those immediately referable to or connected with the delusions themselves. Mr. Justice Stephen, than whom no one has investigated this difficult corner of law with more philosophical and diligent study, made the following remark as to the unexpected and incalculable influence of insane delusion, in his evidence before the Select Committee on the Homicide Law Amendment Bill:

"Where you have a specific delusion of that kind it shows that the mind itself is so deeply disordered in all kinds of ways, you cannot draw the inference that there was an intention to kill or to do grievous bodily harm from the fact of killing as you

could in other cases:—when you get a man under any definite delusion whatever, for aught you can tell the workings of his mind may be such that the act which to you appears to be murder, appears to him in quite another light; and if you read books which give accounts of the workings of the minds of mad people, you will find that directly you get a definite delusion set up, the process of the mind is vitiated as well as the mere result. The delusion runs through everything."

This undoubtedly is true of most delusions, and would especially be true of fixed delusions of persecution accompanied by hallucinations of sense and associated with epilepsy, such delusions, in fact, as those of Thomas Banks, which, in the opinion of the Court of Queen's Bench, left the intelligence and the emotions so free and unaffected. But there are delusions and delusions; delusions in the nascent and others in convalescing conditions of mental disease; delusions which indicate "deep disorder of the mind in all kinds of ways," so that "the process of the mind is vitiated as well as the result;" and contrasted with them there are delusions which scarcely affect the mind beyond their own range, and which, weak and changeable, cannot have the import attached to them in the above quotation. And it is worthy of remark that the Judges sometimes employ this term "delusion" to indicate disturbance of thought arising from insanity, without any particular belief in imaginary facts: thus Lord Penzance, in his judgment, Smith *v.* Tebbitt: "It is no doubt true that mental disease is always accompanied by the exhibition of thoughts and ideas that are false and unfounded, and may properly be called 'delusive.' But the question of insanity and the question of 'delusions' is really one and the same. The *only* delusions which prove insanity are *insane delusions*—and the broad inquiry into mental health or disease cannot in all cases be either narrowed or determined by any previous or substituted inquiry into the existence of what are called delusions."

Chief Justice Cockburn, however, did not adopt this loose interpretation of a term, whose definite meaning it is of the utmost importance to fix with all possible exactness. We have seen that in his attack on the Hopwood will he admitted that there was no evidence of delusion, although there was abun-

dant evidence of loss of memory and of confusion of thought, extending to complete testamentary incapacity. Yet Mr. Hopwood had ideas respecting his eldest son, whom he had disinherited, which were "false and unfounded," and might therefore, according to Lord Penzance, be called "delusive." If Lord Penzance had turned his dictum the other way first, and said that the only delusions of value as evidence being insane delusions, to prove the existence of such delusions it is needful first to prove the existence of insanity, it would have been more logical, although such delusions might have included those beliefs of the insane, which may by possibility be entertained by ignorant, flighty, and fanciful people who are not insane. Insane delusions, however, are recognised by their accompaniments,—insane history, insane conduct, insane feeling, insane physical symptoms,—and by their character, exhibited by comparing them with like beliefs, with like accompaniments, in other persons who are unquestionably insane, that is to say, sufficiently like to convince a candid and instructed mind that they are essentially alike.

But these reflections, if they are well-founded, tend greatly to weaken the position of the Lord Chief Justice with regard to the possibility of such delusions as those of Thomas Banks existing without "the mind becoming deeply disordered in all kinds of ways, so that you cannot draw any inference as to intention," even so simple an inference as to whether he intended to kill a man or not; more so as to an intention even more complex regarding testamentary dispositions. For, observe, these delusions had all the accompaniments of insanity which we have indicated. There was an insane history, residence in the county lunatic asylum; insane conduct, violent excitement; insane feeling, violent aversion; insane physical symptoms, epileptic fits; and it will be scarcely denied by any candid person conversant with lunatics that these beliefs and their accompaniments resemble the insane beliefs and the other marks of insanity in thousands of persons suffering from general insanity so closely that there could be no possibility of doubting that they were of the same kind and nature.

The Lord Chief Justice proceeds in his judgment to comment with admirable precision and elegance of diction upon the great

moral responsibility involved in the discharge of that duty so largely committed to the individual in this country of disposing of his property after death to whom he will. He recognises the power of mental disease to poison the affections and to pervert the sense of right, and thus to abolish the responsibility which this duty involves; but here again he intervenes to assert the possibility of delusion neither exercising, nor being calculated to exercise, any influence upon the particular disposition of the testator. The great moral responsibility attached to the duty of making a just and righteous will appears to us to increase rather than to weaken the argument in favour of the old rule of law which denied the exercise of so responsible a privilege to any one proved to be of unsound mind.

From this aspect of the question the Chief Justice proceeds to discuss the effect of enfeeblement of memory and defect of mind upon testamentary capacity. On this question the most important judgment he quotes is that delivered by Erskine, for the Judicial Committee of the Privy Council, in Harwood *v.* Baker [3 Moore, P.C.], viz.: "Their Lordships are of opinion that in order to constitute a sound disposing mind, a testator must not only be able to understand that he is by his will giving the whole of his property to one object of his regard, but he must also have the capacity to comprehend the extent of his property, and the nature of the claims of others whom by his will he is excluding from participation in that property," &c., and the Chief Justice asks, "why should not this standard be also applicable to mental unsoundness produced by mental disease? It may be said that the analogy between the two cases is imperfect; that there is an essential difference between unsoundness of mind arising from congenital defect or supervening infirmity, and the perversion of thought and feeling produced by mental disease, the latter being far more likely to give rise to an inofficious will than mere deficiency of mental power. This is no doubt true, but it becomes immaterial on the *hypothesis* that the disorder of the mind has left the faculties, on which the proper exercise of the testamentary power depends, unaffected; and that a rational will, uninfluenced by the mental disorder, has been the result."

It is indeed an hypothesis that disorder of the mind as distinguished from "congenital defect or supervening infirmity can leave those faculties unaffected upon which the proper exercise of the testamentary power depends." What are these important and efficient mental faculties which can thus be left unaffected by insanity? The supposition, even as an hypothesis, is that which we conceive to be the fundamental error of the whole judgment, namely the metaphysical or psychological conception that a man's mind is merely a bundle of diverse faculties more or less independent of each other, some of which may be affected by insanity, while others upon which the proper exercise of the testamentary power depends, remain unaffected. The Chief Justice does not adduce any evidence in support of his conviction upon which the judgment and the new law depends, namely that a man's mind is thus constructed; but while disclaiming metaphysical inquiry with great emphasis, he adduces the most metaphysical of arguments, namely that of consciousness, in support of his views. "Every one must be conscious that the faculties and functions of the mind are various and distinct, as are the powers and functions of our physical organisation." Our only possible reply is that we have no such consciousness, but that on the contrary we are conscious that mind in us is one thing or power with various modes or directions of activity, but with one indecomposite individuality. And we think that the neurological science already acquired and even that which we may aspire to, from the pursuit of the same methods which have won all recent discovery, indicate the oneness of mind as contradistinguished from the localisation and diversity of brain function. In neurology we cannot get beyond or behind impressions, reflections, and recollections of sense, and as often as we venture to distinguish a bit of mind apart from a man's whole mind, it will be found to be merely a bit of sense felt, reflected or recollected. But a man's whole mind, that is to say, in the words of this judgment, "the senses, the instincts, the affections, the passions, the moral qualities, the will, perception, thought, reason, imagination, memory," these are not "so many distinct faculties or functions of the mind," but the tissue of sense-impressions, inherited and acquired,

inextricably woven into one whole state of consciousness which attains to new powers or activities, catalogued under the impressive but often misleading terms above quoted. In conclusion, we are of opinion that the late Chief Justice of the Queen's Bench and the great Judges over whom he presided were right in challenging the extremely narrow legal rule with regard to testamentary incapacity, which they reversed, but that the grounds upon which they acted were fallacious, the argument wrong and misleading, the new rule dangerously lax and wide, and the result in the particular instance to which it was first applied a miscarriage of justice. In the early days of the old rule, when an insane man was said to be unable to make a will simply *quia mente caret,* insanity covered but a very moderate portion of the wide field over which it has since been extended. An insane man then almost invariably indicated a person about whose testamentary incapacity there could be no question. But when the same strict rule came to be applied to new forms and degrees of insanity, it was inapplicable, and indeed it then became that very fallacy which we have indicated as the error of those who argue that every insane man ought to be exempt from responsibility for crime; the fallacy of arguing *a dicto secundum quid ad dictum simpliciter.*

There being in all forms of insanity infinite degrees of mildness and severity, it became obviously unjust to apply that rule to the mild and slight degrees of mental disease and defect which was only applicable to degrees of greater intensity. The question of course was always essentially one of motive, that is to say ethical, or, if you like, psychological; but concurrently there was one, we will not say for the medical man, but for any man who diligently studies all the mental qualities and conditions of the human being. This the authors of the old legal rule could not do, because degrees of insanity were scarcely recognised in those days. This also the authors of the new rule have failed to do, because they adopted a metaphysical conception which misled them. What legal rule may eventually be devised and upheld, which shall delineate fairly and fully the characters and degrees of mental diseases which shall, and those which shall not, carry with them testamentary incapacity, must depend upon the attainment

of a better knowledge both legal and mental, and of a more intimate concurrence between equity and science. Whatever the rule may be, however, its application must not unfrequently "involve considerable difficulty and require much nicety of discrimination," as the Chief Justice admitted of inquiries under his own rule. The manner, however, in which such difficulties may be overcome will surely not be by metaphysical or psychological discussion, but by the painstaking method of comparison described by Lord Penzance [Smith *v.* Tebbitt], first, by the comparison which common men make of the words and deeds, as indicating the thoughts of the testator, with the standard of sanity they bear in their own minds; and secondly, the more instructive comparison which those who are conversant with the insane can make between the sayings and doings of the testator with the sayings and doings of those who are undoubtedly insane to an intestamentary degree. And it is, as Lord Penzance argues, by this double comparison of the mind of the testator with the sane mind of the Court, and with the insane mind as it is known and described by competent observers, by which that nicety of discrimination which these inquiries demand may be attained.

We have been led into a longer criticism of this famous judgment than we had intended or expected. Its intrinsic importance, which cannot easily be overestimated, conjoined with the profound respect we entertain for the memory of its author, has forbidden us from expressing our objections with less consideration and amplitude. This judgment in Banks *v.* Goodfellow, full of learning and research as it is, covers much of the ground of insanity in its civil relations, and indeed the Chief Justice himself informed the writer of these pages that he considered this judgment as the full expression of his opinions upon the whole subject. That these opinions are original, and constitute an entirely new law upon the subject, is a sufficient justification, if one were necessary, for subjecting them to a free criticism, which, so far as we know, has not before been done.

THE DIFFERENTIAL DIAGNOSIS OF PARALYSIS.[1]

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Paralysis is a symptom, not a disease. In every case, therefore, in which it comes before us, we must endeavour to ascertain its cause.

The following are the steps in the inquiry :—

1. Is the paralysis genuine?
2. If genuine, is it functional or organic?
3. If organic, what is the position, extent, and pathological character of the lesion?

Step No. 1.—*Is the paralysis genuine?*

The great characteristic of genuine paralysis is "defective or abolished muscular contraction, *while an effort of the will remains.*" (Erb.) In feigned (sham) paralysis there is defective or abolished muscular contraction, because the supposed patient *does not choose to make* an effort of the will. The recognition of feigned paralysis, when the impostor is well up to his trade, may be a matter of great difficulty. The chief points on which reliance is to be placed in making the diagnosis are:

A. *Negative Evidence.*—1. The fact that there are no signs nor symptoms of organic disease.

Feigned paralysis *is to be suspected* when:

[1] A Lecture delivered in the Extra-Academical School of Medicine, Edinburgh, during the summer session 1880, and read as a paper before the Medico-Chirurgical Society of Edinburgh, January 5th, 1881.

(*a*) The condition of the muscles, as regards their nutrition, irritability (mechanical and electrical), tonicity, the condition of the reflexes, &c., is quite normal.

(*b*) There are no associated nerve symptoms; particularly, (1) No symptoms of hysteria, in which condition paralysis of a functional character often occurs. (2) When there is no evidence of mental derangement. (In some cases of mental disease the patient does not or will not move a limb or limbs, but in such cases there is no intention to deceive; the paralysis is not therefore *feigned* in the sense in which I have used the term.)

(*c*) There are none of the conditions present which predispose to or give rise to paralysis, such as disease of the heart, arteries, kidneys.[1]

B. *Positive Evidence.*—If, in addition to the negative facts already mentioned, there is any positive evidence in favour of imposture, *then and then only* is a diagnosis of feigned paralysis justifiable.

The positive facts are:

1. The presence of any anomalous symptoms, such as:

(*a*) Irregular distribution of the paralysis. In cases of hemiplegia, the fact that the arm suffers less than the leg,[2] or that the arm on one side and the leg on the other are affected.

(*b*) Any peculiarity in the paralysed parts. The circumstance that in walking the hemiplegia patient does not circumduct the leg, though not absolute, is of some weight.[3]

[1] Impostors may of course have heart, kidney, or arterial disease. The converse proposition, therefore, that all persons who have heart, kidney, or arterial disease, and who manifest symptoms of paralysis, are impostors, is obviously not true.

[2] In some genuine cases the arm suffers less than the leg, but this is so rare that, if taken in conjunction with the negative facts, mentioned above, it would be very strong evidence indeed in favour of imposture.

[3] In some cases of genuine organic paralysis there is no circumduction of the leg. In functional cases, too, the leg is not circumducted; indeed, according to Todd, this is one of the points of distinction between organic and hysterical hemiplegia.

The fact mentioned by Todd that, in cases of feigned paralysis, when the patient is told to stoop forwards and pick up something from the ground, the paralysed arm is kept fixed to the side instead of falling forwards of its own weight would be conclusive. (In cases of genuine paralysis the arm may be kept fixed to the side by rigidity [contracture]; but in such cases the other facts, especially the condition of the deep reflexes, are so distinctive as to be quite unmistakable.)

2. Any peculiarity in the history, mode of onset of the attack, &c. The fact, for example, that the paralysis followed a railway collision, if coupled with negative evidence, is of some weight in favour of imposture.

3. The fact that the patient has something to gain by the imposture. That he is claiming damages from a railway company; that he has no fixed abode, that he has been in and out of many hospitals, that he is, in short, a "hospital bird" trying to keep a roof over his head by his deceit.

Simulated, but not sham, paralysis.

Paralysis may be simulated when there is no intention on the part of the patient to deceive, as in:

(1) Those cases of mental disease already referred to (see page 28). Under this head we may perhaps include some cases of hysterical paralysis.

(2) In cases of insensibility (coma), it is sometimes very difficult to say whether the patient is paralysed or not.

In the great majority of cases of coma if there is any paralysis it is unilateral (hemiplegia). In such cases a careful comparison of the muscles on the two sides of the body is to be made, as regards:

(*a*) *The condition of the reflexes.*—In cerebral paralysis (hemiplegia) the superficial reflexes are abolished, and the deep reflexes increased on the paralysed side. (The increase of the deep reflexes is usually not apparent until the stage of coma passes off.)

(*b*) *The tonicity of the muscles.*—The paralysed muscles are usually softer and more relaxed, fall with a dead weight, &c. In the case of the face the paralysed cheek is blown

out during expiration, saliva flows from the angle of the mouth on the paralysed side, &c. In the face, too, where the symmetry of the features depends upon the equality of action of two opposing sets of muscles, we can usually detect some drawing of the features to the sound (opposite) side. The presence of (paralytic) conjugate deviation of the eyes and head would at once, of course, settle the question, for this symptom only occurs in severe cases.

(*c*) The temperature, degree of moisture, colour of the skin, on the two sides. In many cases of hemiplegia alterations in the vaso-motor condition of the paralysed parts are observed. In the earlier stages the temperature on the paralysed side is usually higher, the skin more moist, and the colour deeper, than on the sound side.

(*d*) As regards spontaneous movements.

In many cases of incomplete coma, the patient does make some spontaneous movements, but they are limited to the sound side. (An exception to this statement occurs in the so-called "associated movements," which may occur on the paralysed side. These associated movements are usually observed after the coma has disappeared.)

The fact that *convulsive movements* are limited to one side does not, as is sometimes erroneously thought and stated, prove that the other (the immobile) side is paralysed. In fact, in the great majority of cases, the contrary holds good. In the large majority of cases of unilateral convulsions we should be correct in predicting that, if either side were paralysed, it would be the same (i.e. the convulsed) side.

Paralysis may also be simulated by other genuine affections.

1. *Disorders of co-ordination*, notably Locomotor Ataxy, were, and are, even still, mistaken for paralysis. That the disturbance of motion in such cases (the difficulty of walking in the case of locomotor ataxy) is not due to paralysis is proved by the fact that, the gross force of the muscles is well preserved; in some cases, indeed, in which the muscular development is great, the gross force is much above the normal average.

2. *Painful affections.*—In some cases of rheumatic fever, for example, the parts are instinctively kept as quiet as possible, and such cases may be mistaken for paralysis.[1]

Step No. 2.—The paralysis is genuine.—Is it functional or organic?

By functional paralysis we mean those cases in which the loss of power depends upon changes in the motor nerve-tissue of so slight a character as to baffle all our present means of investigation, in contradistinction to those cases of paralysis which depend upon an obvious organic cause.

Functional paralyses are usually temporary in character; the paralysis often sets in suddenly and as quickly disappears.

Under the head of functional paralyses are included:—hysterical paralysis, epileptiform paralysis, paralysis from idea, malarial paralysis, reflex paralysis, and some others.

Hysterical Paralysis.

Before entering upon the differential diagnosis of hysterical paralysis, it is necessary to insist upon the fact, that all cases of paralysis occurring in hysterical patients are not necessarily hysterical (functional). Hysteria, indeed, is frequently associated with organic disease of the nervous system. The presence of hysteria, or a history of hysterical fits, is only, at the most, corroborative evidence. A diagnosis of hysterical paralysis, even when the patient is hysterical, should never, then, be made unless the most scrupulous examination has failed to detect the signs and symptoms of organic disease.[2]

The differential diagnosis of hysterical paralysis is sometimes very difficult, and, since it is a matter of the first importance, the points on which reliance is to be placed in solving the problem will be given in some detail.

A. *Negative Evidence.*

1. The fact that there is nothing in the condition of the

[1] It must be remembered that in some of these rheumatic cases a true paralysis does occur.

[2] This of course raises the question as to what is positive evidence of organic disease—a question which is not yet in every respect absolutely settled. (See remarks on the ankle-clonus, page 32.)

paralysed muscles distinctive of organic paralysis (no "reaction of degeneration," no "rapid atrophy").

In cases of hysterical paralysis the nutrition of the muscles is usually well preserved. This is certainly the fact in the earlier stages; in the latter period of long-continued cases there is atrophy, but it is not more than can be accounted for by disease.

The *irritability* (mechanical and electrical) of the muscles is normal, or possibly slightly increased, but (in the earlier stages) not markedly decreased.

The *tonicity* of the muscles is normal or increased. Sometimes there is rigidity (a spastic condition). In the later stages of chronic cases the muscles may be flaccid.

The condition of the reflexes.—In two cases which I have recently had an opportunity of examining, the skin reflexes were abolished, while the deep reflexes were increased. In both of these cases the ankle-clonus was well marked. On this point, therefore, I differ from Dr. Gowers, who, in his recent most admirable book on the diagnosis of the spinal cord, states that, the presence of the ankle-clonus is proof positive of organic disease. Professor Charcot and Dr. Buzzard have also observed the ankle-clonus in hysterical paralysis.

It will be gathered from this description that, in my opinion, the condition of the muscles in hysterical paralysis (hysterical hemiplegia) differs in no way from the condition of the muscles in many cases of organic cerebral paralysis. The presence of "rapid atrophy" and the "reaction of degeneration," would, of course, prove that the paralysis was organic, and that it was due to a lesion of the nerve trunk or nerve nucleus.

2. That there is no evidence of such disease of the nervous system as might cause, or as is usually associated with, organic paralysis. A careful and detailed examination of the other parts of the nervous system must, therefore, be made in cases of supposed hysterical, as indeed in all cases of paralysis.

It is specially important to note the condition of the optic discs. In some cases of paralysis and hemi-anæsthesia, the

conditions of the optic discs may be the only means of arriving at a positive conclusion.

3. That there is no organic disease of the heart, arteries, or kidneys—conditions which predispose to vascular rupture, embolism and thrombosis, and hence to organic paralysis.

B. *Positive Evidence.*

1. The fact that the patient is a female, generally young; and that she presents symptoms and signs of hysteria—such as emotional tendencies, globus hystericus, tenderness on pressure over the ovarian region, a history of recent or past hysterical fits, &c.

The appearance of the face in cases of hysterical paralysis is often characteristic, but difficult to describe. According to Todd the *facies hysterica* is as follows: "An expression characterised by a remarkable depth and prominent fulness, with more or less thickness of the upper lip, and of a peculiar drooping of the upper eyelid."

2. *Irregular Distribution and Character of the Paralysis.*

There may be nothing peculiar in the distribution of the paralysis in hysteria. In many cases, however, it is irregular; the leg affected more than the arm in hemiplegic cases; one arm and the opposite leg; part of a limb only. In hysterical hemiplegia the face and tongue are very rarely involved. In hysterical hemiplegia, too, the leg is dragged as a dead weight, and is not circumducted. Todd laid great stress upon this point, as the following quotation shows:—"But I would particularly call your attention to the peculiar character of the movement of the paralysed leg when the patient walks, which, in my opinion, is characteristic of the hysterical affection. If you look at a person labouring under ordinary hemiplegia from some organic lesion of the brain, you will perceive that, in walking he uses a particular gait to bring forward the paralysed leg; he first throws the trunk to the opposite side, and rests its entire weight on the sound limb; and then, by an action of circumduction, he throws forward the paralysed leg, making the foot describe an arc of a circle. Our patient, however, does not walk in this way; she drags the paralysed limb

after her, as if it were a piece of inanimate matter, and uses no action of circumduction, nor effort of any kind to lift it from the ground; the foot sweeps the ground as she walks. This I believe to be characteristic of the hysterical form of paralysis."[1]

The *onset* in hysterical cases is often quite sudden, the paralysis appearing abruptly after an hysterical fit or some emotional disturbance. This fact is not of very great value, for organic paralysis also very commonly sets in suddenly, and the exciting cause may be a fright, or other emotional disturbance.

The *course* in hysterical paralysis is very variable. Usually the duration is short, the case terminating suddenly in complete recovery. In other cases the paralysis lasts for years.

Sudden improvements, then, and sudden relapses are characteristic of hysterical paralysis. In some cases of cerebro-spinal sclerosis (sclérose en plaques) the same fact—sudden improvement and sudden relapse—is observed. I have known such a case, for this reason, thought to be hysterical.

In cases of hysterical paralysis associated with contracture, the paralysis and rigidity appear together quite suddenly, differing in this respect from organic contracture, i.e. the late rigidity of Todd. The contracture, too, in hysterical cases, is apt to involve the leg as much as, or more than, the arm. In organic cases (hemiplegia with contracture) the rigidity is often limited to the arm, and never involves the leg alone. In hysterical cases where the contracture involves the leg, the toes are pointed, and the foot somewhat inverted.

According to Charcot, chloroform relaxes hysterical, but not organic contracture. This statement obviously only applies to chronic cases in which the organic contracture is associated with secondary (cirrhotic) changes in the affected muscles.

3. *The Condition of the Sensory Nerve Apparatus.*

At the commencement of organic cases there is generally more or less disturbance of sensibility (anæsthesia), but in the great majority of cases the anæsthesia soon passes off—much more rapidly than the paralysis. In organic cases there is seldom complete hemi-anæsthesia. (An exception to this occurs in those cases in which a lesion involves the posterior part of the *internal capsule* or adjacent parts. In those cases

[1] 'Clinical Lectures on Paralysis,' p. 20.

the motor paralysis is often slight, for the simple reason that, the lesion is not sufficiently extensive to destroy the motor fibres too.)

In hysterical cases, on the contrary, there is usually great disturbance of sensibility. In some, profound anæsthesia, often strictly limited to, but involving all the parts, including the special senses, on one side of the body. In others, sensibility to pain is completely abolished, but sensibility to touch remains. Again, there may be hyperæsthesia, the slightest touch producing complaints of pain and suffering.

4. *The condition of the bladder and urine.*—In hysterical paralysis, retention requiring the use of the catheter is often a prominent symptom. It usually continues so long as the instrument is passed by the House Surgeon, but often yields when his place is taken by a female nurse. The *urine* in hysterical cases, especially after a "fit" or other emotional manifestation, is pale, limpid, and very abundant. In some cases there is ischuria.

5. *The condition of the uterus and ovaries.*—In hysterical cases there is frequently some disorder of the uterine and ovarian functions. Tenderness on pressure over the ovaries is sometimes observed. Slight pressure, according to Professor Charcot, induces an hysterical fit, while firm pressure arrests it. These effects of pressure are not, however, as a rule, seen in the hysterical cases met with in this country.

6. *The previous history.*—A history of previous hysteria, and especially of previous attacks of paralysis which have been rapidly and completely recovered from.

Epileptic Paralysis.

Epileptiform paralysis is a functional paralysis. It occurs in those cases of epilepsy which depend upon the presence of a "coarse" (cortical) lesion, and is rarely, if ever, seen after the convulsions of the so-called "genuine" or "idiopathic" disease. The paralysis is limited to the muscles which were convulsed (or most convulsed); and, since "coarse" cortical lesions are generally unilateral, or limited in extent, the resulting paralysis and convulsions are unilateral, and often limited to one limb, to one group of muscles, or even to one muscle.

The paralysis is supposed to be due to the exhaustion of grey matter (motor nerve cells of the cortex) which follows the excessive and violent, or, as Dr. Hughlings-Jackson expressively puts it, "brutal," discharge which causes the convulsion. Granting, as I do, that this explanation is probably true, it is a remarkable fact that paralysis does not occur after the convulsions of the *idiopathic* disease, in which the discharge, as measured by the severity of the spasms, is very violent.[1]

Epileptic paralysis is quite temporary, passing off after a few hours or days at the most.

Organic paralysis may be associated with convulsions. In some cases of cerebral hæmorrhage the attack is ushered in by convulsions and followed by paralysis. Paralysis and convulsions are also observed in some cases of embolism. It is, therefore, a most important question to decide whether in any given case of convulsions and paralysis, the loss of motor power is of the temporary (functional) kind, or whether it is due to an organic lesion, i.e. permanent. If we can decide in favour of the functional variety, we are able to give great mental comfort to the patient and to his friends; for paralysis is an objective symptom, which is, naturally, regarded by the laity as most ominous. In cases of epileptic paralysis, motor power is, as I have already stated, quickly regained, and under appropriate treatment (iodide of potassium in full doses) the general condition of the patient often improves in a remarkable degree. Great credit and reputation are often, therefore, to be made by the satisfactory recognition and successful treatment of such cases.

The points on which a diagnosis of epileptiform paralysis is to be based are the following:—

1. The fact that the paralysis *followed* a fit (epileptiform convulsion), presenting the characteristic features of the convulsions which result from the presence of a "coarse" cortical lesion:

(*a*) The spasms have a local commencement, and are often limited to one set of muscles, or to the muscles

[1] I have attempted to explain this fact by supposing that there is a difference in the process of discharge in the two cases. See 'Edinburgh Medical Journal,' March, 1881, page 831.

on one side of the body. In some cases the spasms after commencing locally become general (bilateral).

(*b*) The attack is not attended with loss of consciousness, or rather that, so long as the spasms are local, there is no loss of consciousness; for, when the spasm becomes general, consciousness is usually lost, just as in a typical epileptic fit.

2. The fact that the paralysed muscles are those which were most and first convulsed. This is all-important, for it shows that the conducting fibres from the centres to the muscles are sound, and that the lesion is situated in the discharging centre itself. It is not, of course, always possible to say from personal observation which muscles were convulsed. We may therefore have to rely upon the statement of the patient or his friends for information, and such information cannot unfortunately be always implicitly relied upon.

We have now excluded hæmorrhagic apoplexy. (In a case of cerebral hæmorrhage sufficiently severe to be attended with convulsions, consciousness is profoundly affected for some time. The spasms do not recur, or if they do recur, they do not affect the paralysed muscles. There are the other features of a severe apoplectic attack.) And we have to determine whether the paralysis depends upon embolism or thrombosis, or whether it is epileptic, i.e. functional; for in embolism and thrombosis convulsions, unattended by loss of consciousness, may be present in addition to the paralysis, the anæmia or subsequent softening being the common cause of both conditions.

The differential diagnosis of a "coarse" cortical lesion, i.e. of such a lesion as we know by experience is the cause of epileptiform paralysis, on the one hand, and of an embolism or thrombosis on the other, is to be decided by attention to the following additional points:

1. *The presence of the symptoms and signs of a "coarse" cortical lesion.*

(*a*) Headache, usually severe, frequently localised, often paroxysmal, and in such cases sometimes associated with tenderness on skull percussion.

(*b*) Vomiting of a purposeless (cerebral) character.

(*c*) Double optic neuritis or optic atrophy. This symptom is of the greatest importance, for it is present in the majority of cases of "coarse" lesion (tumour), but does not occur in embolism or thrombosis.[1]

2. *The history and mode of commencement of the attack.*—In embolism the convulsions and paralysis occur abruptly, and there may have been no previous cerebral symptoms. In thrombosis there is sometimes a history of vertigo, headache, and evidence of slight sensory and motor disturbance.

In the case of a "coarse" lesion (tumour) there is almost invariably a history of previous cerebral symptoms (i.e. the occurrence of headache, &c., before the attack of convulsions and paralysis).

3. *The associated conditions.*—In the case of a "*coarse*" *lesion* there are often associated lesions of a similar nature (syphilitic, tubercular, cancerous, &c., as the case may be). In embolism there is the presence of cardial or arterial (aortic) disease. The occurrence of the attack during pregnancy, &c. *In thrombosis* the arterial system is diseased, the superficial arteries are usually atheromatous, the patient is, as a rule, past middle age. (In syphilitic cases there may be no evident disease of the superficial vessels, and the patient may be young.)

4. *The effects of treatment.* Epileptiform paralysis speedily disappears; the paralysis due to embolism and thrombosis

[1] In cases of limited meningitis of the convexity, we have a "coarse" lesion, which is usually unattended with optic neuritis. There is fortunately not much difficulty in deciding between such a lesion on the one hand, and embolism or thrombosis on the other. Localised meningitis is either traumatic or tubercular. In the former case the history, in combination with the symptoms, is sufficient to decide the point. In tubercular cases the question is decided by:

1. *The character of the symptoms.*
   (*a*) Headache, usually a prominent symptom in meningitis; often absent in embolism and (though less frequently so) in thrombosis.
   (*b*) Vomiting. Much more frequent in meningitis.
   (*c*) The condition of the temperature. More usually elevated in meningitis than in embolism.
2. *The mode of commencement of the attack.* See text, page 40 (2).
3. *The associated conditions.* See text, page 40 (3).

Dr. Stephen Mackenzie has reported two cases of embolism in which there was double optic neuritis; but this is so rare that the general statement in the text may be safely relied upon.

remains. The general symptoms, too, in many cases of cerebral tumour (syphilitic cases) improve rapidly under appropriate treatment (full doses of iodide of potassium).

Paralysis depending upon Idea.

Under the term "Paralysis depending on idea," Dr. Russell Reynolds has described certain cases of paralysis depending upon imagination. In these cases there is no intention to deceive; the patients really believe that they are the victims of serious organic disease. Cases of this sort are quite distinct from ordinary cases of hysterical paralysis and from ordinary cases of hypochondriasis. The patients are for the most part of a highly, and often very active, mental nervous temperament; their general health is usually below par, but they do not, so far as my limited experience goes, exhibit the usual symptoms of hysteria. The fixed belief that they are paralysed; and the concentrated attention which the affected parts receive, together with the effect, which is produced on the mind by such a state of matters, induces functional disturbances often of a striking character. Startings, twitchings, and fibrillary tremors, occur in the limbs and other parts of the body. Aching muscular (myalgic) pains are common. The heart is easily excited; the patient suffers from attacks of palpitation. Exertion or mental excitement is followed by a feeling of exhaustion and fatigue. The patient becomes impressed with the idea that he is unable to do anything; that he is paralysed, &c. There is often sleeplessness and restlessness. The patient wakes up towards the early morning hours, and rises unrefreshed. The stomach may be deranged; constipation is common. On physical examination the reflexes—superficial and deep—are generally found to be exaggerated, the affected parts (muscles supposed to be paralysed) are usually soft and flabby; in chronic cases there may be considerable atrophy, but it is not limited to special muscles or groups of muscles; a point of importance, for, in some cases which have come under my notice, the diagnosis lay between this condition and progressive muscular atrophy. *Sensibility* is not affected. The *bladder* is healthy; the bowels, as already mentioned, are usually constipated.

The loss of motor power is never complete, and often presents anomalous characters; for example, the patient, who can neither stand nor walk, will move the legs in any direction when in bed, &c.

This condition often lasts for a long time, it may be years; and is sometimes most difficult to cure.

The diagnosis is to be made by attention to the following points:—

1. *The condition of the paralysed parts.*—There is no positive evidence of organic paralysis.

2. *The mental condition and temperament of the patient.*—The fact that the patient's mind is concentrated on his condition; that he is always thinking of his symptoms; examining his limbs, &c.; and that he is thoroughly impressed with the idea that he is the subject of serious organic disease.

3. *The condition of the reflexes.*—The increased reflex excitability, and the fact that it is general, and is not confined to the paralysed parts, is, so far as my limited observation enables me to judge, a diagnostic point of some value.

4. The fact that the startings, jerkings, and fibrillary twitchings are not limited to special muscles, and that the muscles in which they occur do not present any special atrophy.

5. The fact that there is no affection of the bladder or rectum.

6. *The history and progress of the case.*—The fact that the case remains stationary for years, and especially that no marked alterations in the condition of the paralysed parts appear.

In some cases there is a family history of genuine disease of a similar kind. This fact, of course, cuts both ways; for, on the one hand, we know that nerve affections are frequently hereditary, on the other, it must always be remembered that persons of a susceptible nervous temperament, who are brought in contact with disease, are very apt to imagine that they are themselves the subjects of it.

7. *The effects of treatment.*—A favourable opinion, confidently expressed, and appropriate treatment, sometimes effect rapid and remarkable improvement.

Malarial Paralysis.

Temporary paralysis sometimes occurs as the result of malarial infection. The pathology of the condition is unknown. Cases of this sort are rare, even in malarial districts, and no case has come under my personal observation. The characteristic feature of the paralysis is that it is intermittent, and that it occurs at regular intervals, just as the ordinary febrile paroxysms (quotidian, tertian, &c.) do.

The diagnosis is founded upon:

1. The absence of the signs and symptoms of organic disease.

2. The intermittent character of the paralysis.

3. The fact that the paralysis is cured by antimalarial remedies (quinine).

Reflex Paralysis.

Functional paralysis occasionally results from peripheral irritation acting reflexly. The source of the irritation is usually in the bladder or urethra.

Brown-Séquard supposes that the immediate cause of the paralysis is anæmia of the cord, and that this is due to spasmodic contraction of the vessels of the cord produced reflexly. In many cases of so-called reflex (urinary) paralysis there are organic changes in the cord of an inflammatory character. In some of these cases, probably the majority, the inflammatory changes result from direct extension to the cord of similar conditions in the distant (peripheral) parts. The extension may take place along the nerves (neuritis ascendens) or through the blood-vessels.

In some cases organic lesions have been found in the cord, when the intermediate parts have appeared to be healthy, and it is supposed by Benedict and others that, organic spinal lesions can actually be induced reflexly, independently of direct extension.

The cases in which there is an organic lesion of the cord cannot correctly be termed *reflex*, if we understand, as I do by that term, *a functional paralysis due to peripheral irritation acting reflexly.*

True reflex paralysis is undoubtedly rare, but that it does

occasionally occur in man seems beyond dispute; and that it can be induced in the lower animals seems proved by the remarkable chloroform experiments which Brown-Séquard has lately published. The diagnosis of reflex paralysis is always hazardous, and should only be made when:

1*stly*. There is no evidence of organic disease.

2*ndly*. There is a manifest source of peripheral irritation.

3*rdly*, and *chiefly*. The removal of that irritation is followed by the disappearance of the paralysis.

Temporary paralysis due to Vasomotor changes in the cord are sometimes met with, and are clearly functional in character.

Step No. 3.—The paralysis is organic, what is the position and extent of the lesion (anatomical diagnosis), *and what is its character* (pathological diagnosis)?

In seeking to determine the position and extent of the lesion, we must, in the first place, decide whether the paralysis is peripheral, spinal, or cerebral (including under that term pontine and medullary paralyses), and having determined this point we must, in the second place, endeavour to ascertain the particular part of the nerve-trunk, spinal cord, or brain, respectively, which is affected.

The points to which attention is to be directed in making the anatomical diagnosis are:

1. The exact distribution of the paralysis.
2. The condition of the paralysed muscles, especially as regards "rapid atrophy" and the "reaction of degeneration."
3. The condition of the reflexes, superficial and deep.
4. The condition of sensibility.
5. The associated nerve symptoms—more particularly derangement of parts indicating lesions of any particular part of the nervous system (localising spinal and cerebral symptoms).
6. The presence of any obvious cause, such, for example, as a traumatic injury of the skull, spine, or nerve-trunk.

ON THE CAUSE OF THE MOVEMENTS OF ORDINARY RESPIRATION. ARE THESE MOVEMENTS REFLEX?

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THE movements of ordinary respiration, which begin at birth and continue uninterruptedly throughout the life of man and of all warm-blooded animals under normal conditions, have an exciting cause. This cause, whatever it may be, does not operate during the period of intra-uterine existence; and it is well known to physiologists that the part which the lungs play after birth in the introduction of oxygen is performed in the fœtus by the placenta. If the umbilical vessels be compressed in a living animal, the fœtus will soon begin to make respiratory efforts; and this even at an early stage of development. This interesting experiment is by no means new, and it is one that I have often repeated. Another very instructive observation, illustrating the same principle in adult animals, was made in 1664 by the celebrated Robert Hook. In this experiment, it was ascertained that, if air be efficiently supplied to the lungs of a living animal by artificial respiration, as by fixing a bellows in the trachea and regularly inflating the lungs at short intervals with fresh air, respiratory efforts will soon cease, and the animal will remain perfectly quiet so long as the artificial supply of air is properly maintained.

In the first of the experiments above-mentioned, movements are apparently excited because the respiratory function of the placenta is interrupted; in the second, the normal respiratory movements are arrested, as it seems, because the exciting cause of these movements is temporarily removed. The explanation of the phenomena observed in both experi-

ments involves the assumption of the existence of some exciting cause or of a demand on the part of the system which secures, in the adult animal, the regular and periodic introduction of fresh air into the lungs. In normal, tranquil respiration, this cause operates independently of sensation. When there is any deficiency in the supply of fresh air, one evinces a sense of respiratory difficulty, or it may be exaggerated to the point of a feeling of impending suffocation. This sense, both normal and exaggerated, might properly be described as the sense of want of air, or, as it is called by the French, *besoin de respirer*. Many years ago, about 1809, Legallois showed that animals instantly ceased to breathe when the medulla oblongata was destroyed. In 1823 and 1827 Flourens clearly defined a certain portion of the medulla oblongata, near the origin of the pneumogastric nerves, as the respiratory nervous centre. Their experiments and those made subsequently by other physiologists demonstrated conclusively that a nerve-centre situated in the medulla oblongata is the only part capable of appreciating the sense of want of air. When this part is destroyed, respiratory movements instantly cease, for the simple reason that the nerve-centre, which alone is capable of receiving the impression due to want of air, is destroyed. About 1833 the attention of physiologists was directed by Marshall Hall to what are now known as reflex phenomena. He regarded the respiratory movements as reflex, depending upon an impression conveyed to the medulla oblongata through the pneumogastric nerves, received by the medulla oblongata and reflected back through certain motor nerves to the muscles of inspiration.

This brief historical sketch, in which only the most important of the many experiments made on the subject under consideration have been mentioned, serves to show the state of physiological knowledge up to a time a few years later than the observations of Marshall Hall.

In following out the question of the cause of the respiratory movements, it is necessary to appreciate as exactly as possible the mechanism of the appropriation of oxygen by the system.

With each inspiratory act, about twenty cubic inches of fresh air are taken into the respiratory organs to replace about

the same quantity of vitiated air expelled in expiration. The air thus introduced is lighter than the air contained in the deeper part of the lungs, the latter being charged with carbonic acid. It is evident, however, that little if any part of the twenty cubic inches of fresh air can immediately reach the air-cells, in which the interchange of gases between the air and the blood actually takes place, for the lungs ordinarily contain about two hundred cubic inches; but, in accordance with the law of diffusion of gases, there is a constant progression of the air, laden with carbonic acid, from the deeper parts of the lungs toward the trachea, and an equally constant penetration of the fresh air toward the air-cells. In this way, although the fresh air is introduced and the vitiated air is expelled intermittently about eighteen times per minute, the actual contents of the air-cells probably present a pretty constant composition as regards oxygen and carbonic acid; the supply of oxygen being maintained by the repeated inhalation of fresh air, and the blood, in its passage through the pulmonary capillaries, constantly giving off carbonic acid. It is only when the supply of oxygen is deficient that one is actually conscious of a sense of want of air.

There is a corresponding regularity in the current of blood through the capillary vessels in the walls of the air-cells. The heart contracts intermittently about seventy times per minute; but the successive charges of blood that are sent by the right ventricle into the pulmonary artery are received by vessels of great elasticity, which, as it were, absorb the intermittent force of the heart, so that the current which passes through the capillary vessels of the lungs is constant and of uniform rapidity. The venous blood thus passing through the lungs is constantly exhaling carbonic acid into the air-cells and receiving oxygen. The oxygen thus taken up by the blood immediately forms a union with the colouring matter of the blood-corpuscles, the blood becomes oxygenated or arterialized, and is distributed to the system through the branches of the aorta. As the blood passes into the capillaries of the general system, the same cause which secures a constant and uniform current of blood through the pulmonary capillaries, viz. the elasticity of the arteries, produces a constant

and uniform flow of blood, which gives up its oxygen to the tissues and passes into the veins, laden with carbonic acid. The lungs simply serve to present oxygen to the blood; and the blood is the vehicle by which oxygen is carried to the tissues; true respiration, however, consists in the appropriation of oxygen by the tissues, and is constantly going on in every highly organised part in the economy.

The theory of Marshall Hall, that respiratory movements are excited by the accumulation of carbonic acid in the lungs, the impression thus produced being conveyed to the medulla oblongata by the pneumogastric nerves, is disproved by the fact that these movements continue, although modified, after section of the pneumogastrics in the neck; and, as early as 1839, John Reid suggested that the sense of want of air was due in a measure to the circulation of venous blood in the medulla oblongata itself. In 1841, Volkmann made a number of experiments, from which he concluded that the respiratory movements were reflex in their character, but were due to the stimulation by carbonic acid of afferent nerves in every part of the body. Without entertaining any doubt with regard to the reflex character of the respiratory movements, I made, in 1861, a series of experiments, in which I endeavoured to show that the sense of want of air was due to want of oxygen in the general system, and not to the stimulation of afferent nerves by carbonic acid. In these experiments, I first showed that in living animals, after the respiratory movements had been arrested by artificial respiration, respiratory efforts began (when artificial respiration was interrupted) only when the blood became dark in the arteries. Recognizing the fact that oxygen can reach the tissues only through the blood, I drained the animal of blood, still keeping the lungs supplied with air, and always succeeded in this way in exciting respiratory movements. The views which I then entertained were still further supported by experiments made by Pflüger, in 1868, who excited respiratory movements in animals by insufflating the lungs with an irrespirable gas, such as pure nitrogen.

Having often repeated my experiments since 1861, frequently as class demonstrations, it occurred to my mind in 1877 that possibly the respiratory movements might be due to some

direct change in the conditions of the medulla oblongata, and I began to entertain doubts with regard to their reflex character. I then undertook a series of experiments which led me to the conclusion that the sense of want of air is due to the want of oxygen in the medulla oblongata itself. The general results of these experiments, the details of which have been published elsewhere, were as follows :—

In a dog brought fully under the influence of ether, artificial respiration was established by means of a bellows fixed in the trachea so completely that all respiratory efforts on the part of the animal ceased. The innominate artery and the left subclavian artery were then exposed so that the vessels could be constricted at will. The irritability of the medulla was tested by interrupting artificial respiration, which was followed by respiratory efforts. Then, artificial respiration having been resumed so that the animal remained perfectly quiet, the great vessels given off from the arch of the aorta were constricted, the artificial respiration being continued. This was invariably followed by violent respiratory efforts, which began in a little more than two minutes after constriction of the vessels and continued until the vessels were freed, when the efforts ceased. No such phenomena followed constriction of the aorta below the arch, which, of course, shut off the blood from all parts of the body except the head and anterior extremities. The experiments of which this is an example were frequently repeated, always with the same results. Of course, if it be assumed that the medulla oblongata is the sole respiratory nerve-centre, it is reasonable to suppose that the occlusion of the vessels given off from the arch of the aorta, which cuts off the supply of oxygenated blood from the medulla, gives rise to a sense of want of air by reason of some change in the conditions of the medulla, which conditions are again changed so soon as blood is allowed to flow again through these vessels.

Reasoning from the facts developed by my own experiments, taken in connection with what is well known and established with regard to the action of the medulla as a respiratory nerve-centre, the following seems to be a satisfactory explanation of the mechanism of the ordinary respiratory acts :—

The left ventricle sends arterial blood received from the lungs to all parts of the system, including the medulla oblongata. The elasticity of the aorta and of its branches gradually extinguishes or absorbs the intermittent force of the heart, so that the blood flows in a steady and continuous stream through the capillaries of the medulla. But, as the tendency of the air in the pulmonary cells is to progressively increase in its proportion of carbonic acid and to diminish in its proportion of oxygen between two respiratory acts, the tendency of the blood coming from the lungs and sent by the left ventricle to the medulla oblongata is to become progressively poorer in oxygen. After about four revolutions of the heart, assuming that the proportion of the beats of the heart to the respiratory acts is four to one, the quantity of oxygen supplied to the medulla oblongata has become so far diminished that there occurs an unconscious sense of want of air, and this excites a new inspiratory act. So it is, in all probability, that the normal rhythmical acts of inspiration are periodically excited; and anything, like violent muscular exercise, that increases the activity of the consumption of oxygen, of necessity increases the number of respirations per minute.

When there occurs any serious interference with the passage of fresh air to the air-cells or an obstruction to the flow of arterial blood to the medulla oblongata, as in certain pulmonary and cardiac diseases, the unconscious sense of want of air is exaggerated until one becomes conscious of pulmonary oppression or impending suffocation. This is simply an exaltation and extension of the normal respiratory sense so that it reaches the true sensory centres, causing a voluntary increase in the number and extent of the respiratory acts. The sense of suffocation, indeed, differs from the normal respiratory sense merely in degree and in the fact that the former operates on the centres of ordinary sensation, while the latter is confined to the medulla oblongata.

When respiration has been so long obstructed that respiratory efforts cease, the medulla rapidly loses its capacity to appreciate the sense of want of air; still, under these circumstances, if the heart continue to beat, artificial respiration, if persisted in so as to restore the supply of arterial blood to the

medulla, will often restore the sensibility of the respiratory nerve-centre, so that finally the respiratory movements will become re-established. Physiologists who are in the habit of administering anæsthetics to animals have frequent occasion to note this fact. A dog, for example, becomes so overpowered by the anæsthetic that the sensibility of the medulla is for the time destroyed and respiration is arrested; the heart, however, continues to act, though its contractions are feeble; but artificial respiration, if kept up efficiently and persistently, will maintain the action of the heart, the respiratory sensibility of the medulla gradually returns, and after a time the respiratory movements are re-established.

Narcotics also may affect the respiratory sensibility of the medulla so that the frequency of the respiratory acts is diminished and they may be arrested; and in such instances it is sometimes possible to revive the respiratory function by artificial insufflation of the lungs. In most cases of suspended respiratory action from any temporary cause, although galvanism, sudden and active stimulation of the surface, &c., may aid in restoration, the main reliance should be upon persistent and efficient artificial respiration.

Are the Normal Respiratory Movements either entirely or in part Reflex, in the Sense in which the term Reflex is ordinarily understood by Physiologists?

I shall leave out of this question various modifications of the respiratory acts, such as coughing, sneezing, &c., and the influence of certain unusual impressions made upon the general surface, as by a cold douche, and restrict the discussion to the phenomena of ordinary respiration. Experiments have shown that the unconscious and automatic movements of respiration in an animal can be abolished by artificially supplying the lungs with fresh air, which has the effect of securing to the capillaries of the medulla oblongata as well as of other parts a sufficient quantity of oxygenated blood. This supply of oxygen through the blood removes the exciting cause of the respiratory movements by abolishing for the time being the sense of want of air. When, however, there is a deficiency in the supply of oxygen to the medulla, this deficiency produces

a sense of want of air, and respiratory efforts follow. The quantity of blood sent to various parts of the body is subject to frequent variations through the influence of the vaso-motor nerves upon the muscular coat of the arteries; in other words, these nerves may modify and regulate local circulation independently of the action of the heart; but the circulation in the medulla oblongata seems to be the physiological gauge of the requirements of the general system for oxygen; and it is by virtue of this property that the medulla is enabled to act as the respiratory nerve-centre. Every vascular part of the body undoubtedly requires oxygen, a proper supply of which is necessary to its nutrition and to the performance of its functions. Normal variations in nutritive or functional conditions may and do involve in certain parts great modifications in the supply of blood, which modifications are regulated by vaso-motor nerves; but these variations in the local circulations do not usually produce impressions capable of modifying the respiratory acts, unless they be attended with an actual increase in the quantity of oxygen consumed in the organism. In case the consumption of oxygen be increased from any cause, the quantity supplied by the lungs must also be increased in order to keep the proportion of oxygen in the arterial blood at the proper standard. The respiratory nerve-centre seems to take cognisance promptly of any deficiency in the supply of oxygen to its substance, and it measures by this deficiency the respiratory demands of the system. With a knowledge of the results of recent experiments, one can hardly imagine that the different parts of the organism, possessing, as they do, such a variety of functions and properties, and subject to such modifications in the supply of blood which they receive, can all be endowed with a common sense capable of being conveyed to the medulla oblongata and there appreciated as the sense of want of air. With the variations to which the circulation in special parts is subjected, it would seem that a confusion of impressions must necessarily result, if these impressions originated in the general system. It is only in the pulmonary structure, indeed, that any such impression could by any possibility arise; but this is not the fact, for the following reasons: Respiratory movements occur when the nervous connection between the

lungs and the medulla has been severed. When the supply of oxygenated blood to the medulla has been cut off, respiratory efforts occur, even though the lungs be fully supplied with air and no deficiency of oxygen or excess of carbonic acid can exist in the air-cells.

Taking into consideration all the facts bearing upon the question, I can come to but one conclusion with regard to the character of the movements of normal, tranquil respiration.

When perfectly normal, and not modified by any unusual physiological conditions, the regular acts of the tranquil respiration are not reflex, at least in the sense in which the term reflex is ordinarily understood by physiologists. As I have said before, there is a constant and uniform current of arterial blood to the medulla oblongata, and there is a progressive diminution in the proportion of oxygen in the blood, between the respiratory acts. After a certain time, the supply of oxygen to the medulla becomes so far reduced that it gives rise to an unconscious sense of want of air, which excites an act of inspiration. This repeats itself regularly from eighteen to twenty times in a minute. Under these conditions, regular respiratory movements are excited by a stimulus generated in the medulla oblongata itself, and depending solely upon the state of the circulation in this part.

Normal Modifications of the Respiratory Movements by Reflex Action.

Although regular movements of respiration may go on without any action that can strictly be called reflex, reasoning with regard to respiration from the phenomena observed in connection with other important functions, one would naturally expect to find the process by which oxygen is introduced into the body subject to modifying and regulating influences operating through the nervous system. The action of the heart is undoubtedly automatic, but the force and frequency of the contractions of this organ are certainly controlled more or less, under physiological conditions, through the nervous system. The connection between aëration of the blood and the circulation is certainly very close; physiological conditions must frequently occur which demand changes in the rapidity

and extent of respiratory acts without involving voluntary effort or even consciousness; and many of these demand the intervention of nervous action aside from the mere development of the sense of want of air in the medulla oblongata. Admitting the truth of my propositions, as regards perfectly tranquil respiration, it is well known that important physiological reflex influences upon the acts of respiration operate through the pneumogastric nerves.

A relatively strong galvanic current applied to the pneumogastric nerves in the neck, or to certain branches of the pneumogastrics (the superior and the inferior laryngeals), will instantly arrest respiration. This action is reflex, as is shown by the fact that galvanisation of the central ends only, of the divided nerves influences respiration. The fact just stated is marked and constant; at the same time, it has been noted that galvanisation of certain other sensory nerves will arrest respiration, although this result is not invariable. When the respiratory movements are completely arrested by galvanisation of the pneumogastrics, it is always the same for the general movements of the animal. On the other hand, "a feeble excitation accelerates the respiration; a more powerful excitation retards it; a very powerful excitation arrests it. These words 'feeble' and 'powerful' having, it is understood, only a relative sense for any one animal, and under certain conditions: what is feeble for one would be powerful for another," &c. (Bert.)

As far as these experimental facts can be applied to the physiology of ordinary respiration, it seems that the nerves, the action of which is brought into play under physiological conditions, must be mainly, if not exclusively, the pneumogastrics. These nerves have their origin at the medulla oblongata; they are distributed to the entire respiratory apparatus, from the larynx to the deepest parts of the lungs, but not to the respiratory muscles, except the intrinsic muscles of the larynx; and they are capable, by reflex action, of exerting a very marked influence over the respiratory movements.

It is important, in this connection, to note another experimental fact with regard to the pneumogastrics. When both of these nerves are divided in the neck, the excitation directly

produced by their section momentarily accelerates the respiratory movements. In animals in which the walls of the larynx are sufficiently rigid to enable the acts of inspiration to be carried on without any serious obstruction, the following phenomena are observed :—For a few seconds, rapidity of the respiratory movements may be increased, but so soon as tranquillity is restored, the number of respirations per minute is very much diminished, falling from sixteen or eighteen to four or five, and the inspiratory acts become unusually profound, and are attended with excessive dilatation of the thorax.

The respiratory phenomena following galvanisation and section of the pneumogastrics in the neck, have never been satisfactorily explained; and certainly such an explanation would throw some light upon the reflex action of these nerves in normal respiration. If, however, my view of the action of the medulla oblongata in tranquil respiration be accepted, a very interesting proposition made by Rosenbach, in 1878, renders it possible to present a theory which, while it may not be entirely satisfactory, is interesting and suggestive, and may be sustained by future experimental observations. Rosenbach advances the idea that the pneumogastrics are the vaso-motor nerves of the medulla oblongata, and that they contain fibres which contract and fibres which dilate the blood-vessels. This is presented merely as an hypothesis, which he "hopes later to be able to establish by experiments."

If it be assumed, for sake of argument, that the pneumogastrics actually contain fibres which regulate the supply of blood to the medulla oblongata, it would be easy to understand how such fibres could so influence the supply of oxygen to the respiratory nerve-centre as to secure a succession of respiratory acts such as would be demanded by the system under different physiological conditions; still, the seat and the exact nature of the impressions giving rise to reflex changes in the calibre of the vessels of the medulla would be a matter of speculation and conjecture.

Applying the suggestion made by Rosenbach to experiments upon the pneumogastrics, the phenomena observed could be explained as follows :—

Relatively feeble galvanisation of the pneumogastrics ac-

celerates respiration by contracting the vessels of supply of the medulla, and thus diminishing the supply of oxygen.

Powerful galvanisation of the pneumogastrics arrests all movements in animals, including the respiratory movements.

When the action of the medulla is removed from the influence of the pneumogastric nerves, as it is after the division of both nerves in the neck, air is taken into the lungs when the deficiency of oxygen in the medulla has reached the point at which the sense of want of air necessarily generates the stimulus sent to the inspiratory muscles. This action is in no sense reflex, and it depends entirely upon the development *de novo* of a stimulation or an irritation in the medulla itself. Under these conditions, the acts of inspiration are abnormally infrequent, and they become excessively prolonged and profound. It is probable that death occurs in a few days after this operation, not alone from abnormal respiratory action, but from a suspension of other important functions which the pneumogastrics have to perform. It is possible, also, that some of the phenomena which are observed in narcotic poisoning—notably a great diminution in the frequency of the respiratory movements—may be due in part to an interference with the respiratory functions of the pneumogastrics.

As far as muscular action in tranquil respiration is concerned, I have thought it necessary to consider only the acts of inspiration; for expiration is produced mainly by the passive contraction of the capacity of the thorax, and by the resiliency of the elastic pulmonary parenchyma succeeding the action of the muscles which enlarge the chest and inflate the lungs.

The results of my experiments, observations, and reflections upon the cause and nature of the movements of normal respiration may be embodied in the following propositions:—

1. The respiratory sense, *besoin de respirer*, sense of want of air, or the stimulus which gives rise to respiratory efforts, is due purely and simply to a deficiency of oxygen in the medulla oblongata, which is the sole respiratory nerve-centre.

2. In perfectly tranquil, uniform, and undisturbed respiration, the regular and successive inspiratory acts may go on in

obedience to successive stimuli, originating *de novo* in the medulla oblongata. Such respiratory movements are not reflex.

3. The frequency and the extent of the normal inspiratory movements are regulated and accommodated to the physiological requirements of the system by reflex action operating through the pneumogastric nerves.

ON SOME POINTS IN THE DIAGNOSIS AND TREATMENT OF BRAIN DISEASE.

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(Concluded from Vol. III. p. 308.)

WHILE therefore our knowledge of the clinical features of thalamus opticus lesions is as yet very fragmentary, we possess a large body of evidence with regard to the distinctive characters of disease in the neighbouring ganglionic mass of the *corpus striatum,* the several portions of which were first clinically differentiated by the observations of Tuerck, Meynert and Charcot, as they had long before been anatomically unravelled by Burdach in his great work on the structure of the brain. The prominence given, and the great interest attaching to the affections of the different portions of the corpus striatum, which are more frequent and practically important than any other localised brain-lesions, have caused the terms of lenticular and caudate nucleus, and external and internal capsule,[1] to become household words in neurological literature.

The term "corpus striatum" is now used in a somewhat different sense by various writers. Most pathologists comprehend by it (*a*) the grey caudate nucleus, Schwanzkern, or intraventricular portion of the ganglion, which comes in sight on laying bare the lateral ventricle; (*b*) the lenticular nucleus, Linsenkern, or extraventricular portion, which is imbedded in the cerebral substance, and therefore not visible from the ven-

[1] Singularly enough, the terms "external and internal capsule" are nowhere to be found in the last edition of 'Quain's Anatomy,' by Sharpey, Allen Thomson and Schäfer. (1878.)

tricle; and (*c*) the white internal capsule, which is situated between the two nuclei, and forms the connecting link between the crus cerebri on the one hand, and the corona radiata and the cortex, of the brain on the other hand. Prof. Dalton,[1] of New York, and others have recently restricted the term corpus striatum to the caudate nucleus or intraventricular portion of the ganglion; while in the following remarks, for the sake of clinical convenience, both nuclei, the internal and external capsule, and the claustrum, will be comprehended under the name of corpus striatum. The claustrum (Vormauer, avant-mur) is that thin layer of grey matter which separates the lenticular nucleus from the convolutions of the island of Reil, the external capsule being a correspondingly thin layer of white fibres on the inner side of the claustrum.

(*a*) *External Capsule and Claustrùm.*—It has long been known that hæmorrhage to a very large amount is apt to occur between the external capsule and the external surface of the lenticular nucleus; and Charcot says that the artery here in fault—one of the lenticula—striated arteries arising from the middle cerebral—is so frequently affected by sclerosis leading to the formation of miliary aneurisms, with subsequent rupture and hæmorrhage, that it almost seems to deserve the name of "cerebral-hæmorrhage-artery." More than forty years ago Gendrin had already called attention to the fact of hæmorrhage occurring more outside than inside the grey nuclei of the corpus striatum; and his observations have since then been extended chiefly by Bouchard, Bourneville, and Duret, and in this country by Dr. Broadbent,[2] who has shown that the effused blood in its further course through the brain-substance performs a kind of natural dissection in the direction of the least resistance. Dr. Broadbent's cases, however valuable in themselves, cannot by any means be taken to afford any information concerning the special functions of the external capsule and claustrum, as has been assumed, for in none of them was the lesion limited to these structures; on the contrary, it was always found to extend to distant parts, generally ploughing

[1] 'BRAIN,' Part X. p. 147.

[2] On Ingravescent Apoplexy. 'Medico-Chirurgical Transactions,' vol. lxix. p. 335. 1876.

up the hemisphere more or less, and bursting into the lateral ventricle. This great extent of the hæmorrhage quite unfits these cases for being utilised for localisation, strictly so called, as for that only limited lesions are suitable. An additional reason for our not making such use of these cases is that all the patients died shortly after the attack. One of them, indeed, survived for three weeks and a half, but the others were carried off within some hours, or, at most, a few days after the seizure. Now recent cases, more especially of hæmorrhage, are most deceptive if employed for the purpose of localisation, inasmuch as at an early period of the disease the symptoms are not exclusively owing to destruction of tissue, but also very greatly to pressure of the clot on neighbouring parts, whereby the circulation in them is altered, and function inhibited. As the clot contracts, these latter symptoms gradually disappear; and about six or eight weeks after the attack those which remain are probably mainly owing to destruction, and then fit for diagnostic purposes. Dr. Broadbent has, in the paper just referred to (p. 358), called special attention to the circumstance that he found hemianæsthesia accompanying the paralysis, not only where the thalamus or the posterior portion of the internal capsule was damaged, but where the hæmorrhage was too far forward for either occurrence. This has led him to think that the French school has referred hemianæsthesia too exclusively to a lesion of the posterior part of the internal capsule, and that that symptom may also be induced by lesion elsewhere. It will be seen from the above considerations, however, that this conclusion as to the causation of hemianæsthesia is not justified by the cases on which Dr. Broadbent has relied for his argument. Had his patients survived the lesion a little longer, the hemianæsthesia might have gradually disappeared, as is not uncommonly seen in similar cases, showing that that symptom was owing not to destruction, but to temporary inhibition merely.

Hæmorrhage localised in the external capsule and claustrum is very rare; and softening limited to these parts does not occur. Temporary crossed hemiplegia would appear to be the symptom corresponding to the above lesion; yet there may be no morbid sign whatever from the commencement, and

it therefore seems impossible to diagnose disease of these structures.

(*b*) *Linsenkern, lenticular nucleus.*—Small areas of softening in this region may remain latent during life; but if the larger part of the nucleus be affected, opposite hemiplegia is the result, which is generally incomplete in character, and transitory in appearance. The recovery of function which appears to be the rule in such cases, is probably owing to compensation being effected by means of the uninjured portions of the linsenkern itself, or of the caudate nucleus. In some of these cases nothing but paralysis of the lower branches of the portio dura has been observed. Hæmorrhage seems never to be strictly limited to the linsenkern, but always to involve portions at least of the internal capsule at the same time; while tumours have been found involving symmetrically the nuclei of both sides, and yet had been totally unsuspected during life (Rondot, Fürstner).

(*c*) *Schwanzkern, caudate nucleus.*—Lesions affecting this ganglion in its entire extent, without encroaching upon other neighbouring structures, have not yet been observed, which is no doubt owing to the peculiar distribution of blood-vessels in this part. Its head alone has occasionally been found softened where there had been no symptoms during life exciting suspicion of such an occurrence. Where portions of the internal capsule have suffered in company with the schwanzkern, crossed incomplete and transitory hemiplegia has been the result. From all this it appears that we are unable to distinguish during life a lesion in one grey nucleus from a similar one in the other; and that, where hemiplegia remains permanent after an apoplectic seizure, the lesion cannot be limited to either of the grey nuclei of the corpus striatum.

(*d*) *Internal Capsule.*—It has been stated that hæmorrhage into, or softening of, the white medullary matter of the internal capsule always causes permanent and incurable crossed hemiplegia; but such is not the case. Here, as elsewhere, the extent of the area of disease is of the greatest consequence, for small lesions may exist even in this important locality without leading to striking symptoms during life. Nevertheless it is a fact that, in the vast majority of cases, somewhat extensive

destruction of this part is followed by crossed hemiplegia of the portio dura and the extremities. The conducting paths for the portio dura and the extremities are probably quite distinct within the internal capsule, as cases are on record where the extremities suffered exclusively, and others where the lower branches of the portio dura suffered exclusively, from disease in this region. The hypoglossus is not so commonly involved as the portio dura; indeed, the tongue appears sometimes quite straight, even in the commencement, and there may be no difficulty of articulation. In other cases the tip of the tongue deviates, and there is dysarthria or anarthria, such as we see it in medullary or pontine lesions. These symptoms, however, frequently vanish in the further progress of the case, from which it may be concluded that they were owing not to destruction, but to inhibition of the function of the cortical or subcortical centres of the hypoglossus, by pressure of the clot. As this latter contracts, the pressure is relieved, and function resumed to a proportionate extent.

That hemiplegia should be more or less permanent from disease of the internal capsule, and temporary from affection of the grey nuclei, is explained by considering the fibres of this capsule as centrifugal in their action, intended to transmit to the extremities and the portio dura the motor influence generated in the cineritious substance of the hemispheres as well as in the caudate and lenticular nucleus. While, therefore, lesion of the lenticular nucleus, for instance, will not prevent transmission of power from the caudate nucleus and the cortex of the brain, destruction of the internal capsule puts an impediment in the way of conduction of any motor power, from whatever source generated. It would therefore follow that only regeneration of nervous fibres in the capsule could, by re-establishing conduction between the centre and the periphery, lead to disappearance of the paralysis in such cases; and this appears, unfortunately, to be a very rare occurrence.

The localisation of the conducting paths for motion and sensation in the different portions of the internal capsule constitutes one of the most brilliant discoveries in this chapter of pathology. According to Charcot and Flechsig, lesions of the internal capsule alone, without any participation of the grey nuclei,

will cause permanent hemiplegia with late rigidity, without permanent affection of sensibility, if limited to the two anterior thirds of the capsule, where this separates the anterior end of the linsenkern from the head of the schwanzkern; while, on the other hand, destruction of the posterior third of the posterior segment of the internal capsule, where this lies between the thalamus opticus and the posterior end of the linsenkern, will cause crossed hemianæsthesia without motor paralysis.

Hemianæsthesia from disease of the internal capsule may be complete and permanent. It then affects not only the entire skin of one half of the body from vertex to toe, but also the mucous membranes of the eye, nose, mouth and tongue, the external auditory meatus, one half of the anus, glans penis, or vagina, as well as the special senses of sight (with negative ophthalmoscopic appearances), of hearing, smell and taste. In other cases it is incomplete, affecting only the skin, and leaving the mucous membranes and special senses intact; or slight in degree, so that sensation is only impaired, but not lost. Where it is complete, the muscular sense and electro-muscular sensibility are likewise absent, so that, while the nerves and muscles respond well to faradisation and galvanisation, no sensation is excited by the electro-muscular contraction; while tendon-reflex excitability is unaffected. In some cases hemianæsthesia disappears after a time, showing that it was not owing to destruction, but to inhibition of function; and similarly in a case where there were at first both hemiplegia and hemianæsthesia, the latter may remain and the former gradually disappear, showing that there was an inhibitory lesion in the anterior, and a destructive one in the posterior part of the capsule.

It is perhaps well to remark that hemianæsthesia of the skin may also occur in lesions of the mesocephale (pons, crus cerebri) and of the corona radiata, so that it cannot be considered as strictly characteristic of internal capsule disease.

Lesions of the *centrum ovale* are as yet little studied, and their symptoms are so manifold in character that in most instances in which they occur, an accurate diagnosis cannot be made during life. Comparatively large portions of this connecting link between the central ganglia and the cortex

may be destroyed by softening or hæmorrhage, without giving rise to any symptoms at all. This appears to be more particularly the case where the occipital, parietal, sphenoidal, and anterior and middle frontal portions are affected. On the other hand, disease in the posterior frontal and posterior central part causes the typical symptoms of corpus striatum paralysis, *i.e.* opposite hemiplegia with paralysis of the lower branches of the portio dura, and sometimes of the hypoglossus. Where the left hemisphere is the seat of it there may also be aphasia, which we are at present unable to distinguish from aphasia owing to destruction of Broca's convolution; and dysarthria which resembles the same symptom from disease of the pons Varolii, medulla, &c. The lesion of aphasia is in the posterior frontal part, close by the third left frontal convolution, and is probably owing to disruption of what Meynert has called the "system of association." Where a small area is affected, there may be only monoplegia. Early and late rigidity in the paralysed muscles is also sometimes found. Tumours invading the centrum ovale may give rise to the general symptoms of increased intracranial pressure, such as vomiting, optic neuritis, headache and epileptiform seizures, without the presence of any regional phenomena enabling us to localise the seat of the disease. It is also worthy of note that destruction of the occipital portion of the centrum ovale has been found associated with acute bedsores on the opposite side.

By lesions of the *cortical substance* of the hemispheres we understand not only those which affect the cineritious substance itself, but also the white medullary matter immediately underlying the same. Of all cerebral convolutions the third left frontal has, since Broca's observations, received the largest share of attention; and a number of typical cases in which there was limited destruction of the same, have rendered its intimate relation to the production of intelligent language absolutely certain. Yet a number of questions connected with this point still await solution. Is this the only centre for language, or are there other portions of the brain which have a close and constant relation to that faculty? What is the part played by the island of Reil and the first left temporal

convolution? How are we to explain some undoubted cases of destructive lesions, not only in the left, but in both third frontal convolutions, in which there was no aphasia? Have the parietal lobes any relation to the faculty of intelligent language, &c.?

As at the present day any opposition to the main features of Broca's theory proceeds only from those who are wilfully blind or disingenuous, the least that the adherents of that doctrine have a right to ask for from their opponents is a thoroughly complete description of the cases brought forward, with the view of disproving the same. Thus it is indispensable to know whether the subjects have been right or left-handed during life; for it is an established fact that, where there is congenital or acquired deficiency of the left hemisphere, the right side of the brain will be trained during infancy for speech, and for the more complex movements of the upper extremity. The omission of this particular renders such cases as those of Simpson, Christison and others, entirely devoid of value. Then, again, it is now well understood that part of the third convolution may be destroyed, and yet its function not be seriously interfered with, if only that portion of it in contact with the operculum—which is situated at the junction of the two branches of the fissure of Sylvius, and covers the convolution of the insula—has remained intact. It is therefore an important reservation to state that language is, in general, *bound to the opercular portion of the third frontal convolution.* For other cases which are apparently contradictory, the bilateral function of the parts in question offers a ready solution. With all these reservations, however, a few cases remain which would seem to show that the function which is in the immense majority of cases bound to the third left frontal may occasionally be undertaken by the insula of Reil, either alone, or in connection with the first left temporal convolution and perhaps the gyrus angularis. A somewhat constant relation of the first left temporal to Kussmaul's "verbal deafness" seems to have been established. Patients of this kind appear either deaf or insane, although they are neither, because they are unable to realise the meaning of spoken words; and this affection is in some instances combined with aphasia, agraphia,

and alexia. It is an interesting fact that in one of Kussmaul's cases verbal deafness was associated with disease in the right first temporal in a man who had been left-handed during life, and in whom therefore congenital deficiency of the left hemisphere could be assumed.

Next in importance to the questions connected with the third frontal and the first temporal is the occurrence of motor paralysis from disease of the convolutions bordering the fissure of Rolando, viz. the anterior and posterior central, and the paracentral lobule. Lussana and Lemoigne have entirely failed to shake the large array of facts which prove such a connection; yet numerous points in this matter are not as yet cleared up. If we accept, for instance, the topography of centres for the tongue, portio dura, and extremities, as given by Charcot and Pitres, it is difficult to explain the occurrence of cases of hemiplegia resembling that which is caused by internal capsule hæmorrhage, and coming on without disease involving the entire region of Rolando, the lesion being limited to a comparatively small area in it. Again, the dissociated or piecemeal appearance of paralysis is not absolutely distinctive for cortical lesions, for occasionally loss of motor power may come on suddenly together with loss of consciousness, in disease of the central convolutions; while it may appear piecemeal, and without affecting the sensorium, in lesions of the pons Varolii, the crus, and the centrum ovale.

The occurrence of signs of motor iritation which is generally believed to be characteristic of lesions in Rolando's region, likewise raises questions which have not as yet been satisfactorily answered. Leaving out of view the presence of late rigidity owing to secondary sclerosis in the pyramidal strands, which may follow cortical lesions just as well as corresponding affections in the centrum ovale, the internal capsule, &c., we are confronted with localised clonic or tonic spasms, and with cortical or Jacksonian epilepsy. Why do these symptoms occur in one set of cases, and not in another apparently exactly similar one? How are they produced? And what is the reason that neither the kind of disease—whether tumour, softening or hæmorrhage—nor its seat in one or the other of the three convolutions in question, nor its invasion of the

cineritious or the underlying medullary matter, seem to have any influence in the production of these phenomena. That their presence or absence follows a definite law cannot be doubted; but much careful clinical as well as pathological work will have to be done before this law can be formulated.

The question of treatment of localised brain-lesions has been generally lost sight of by those most eager to investigate their symptoms and pathology; yet this must for all time to come be the chief point of interest for the practical physician. I will in this place not speak of the attempts which have been made to arrive by trephining at the seat of the disease, as I have elsewhere expressed my disbelief in the future of cerebral surgery.[1] A subtler agent than the surgeon's knife or trephine is wanted for this branch of therapeutics. The chief practical points I wish to submit to the attention of neurologists in this place are two, viz. 1st, the expediency of treating tumours of the brain as if they were syphilitic in origin, even where this cannot be demonstrated; and 2nd, utilisation of the catalytic effects of the constant voltaic current, in certain destructive as well as irritative lesions, directed locally to those portions of the brain where we suspect the seat of the mischief, in accordance with the teachings of pathology and with Ferrier's physiological topography of the cerebral cortex.

I will now relate a case in illustration of my first point, viz., the advisability of antisyphilitic treatment for tumours of the brain:[2]—

CASE 1. *Tumour in the Right Cerebral Hemisphere.*—A merchant, aged 32, married and father of two healthy children, consulted me in April 1878. He had always been in good health until about six months ago, when apparently without any particular cause he began to be seized occasionally with attacks of giddiness and vomiting. This was believed to be owing to congestion of the liver, and treated as such. Headache supervened after a time, his sleep became disturbed, and he appeared sometimes delirious at night. The appetite began

[1] 'Diseases of the Nervous System,' vol. i. p. 45.

[2] A similar case was recently brought before the Medical Society of London, and afterwards published in the 'British Medical Journal,' by Dr. Hughlings-Jackson.

to fail next, and he could not be induced to eat anything substantial. He generally brought up almost directly what little he took. When I first saw him, he was so much emaciated that his friends believed him to be consumptive. He had a sallow complexion, and a careworn countenance. He complained of persistent headache, which was generally of a dull aching character, but sometimes became acute and throbbing, and "made him feel quite silly;" of attacks of giddiness which seemed to take him off his feet, so that he felt as if he was floating in the air; and of greatly impaired memory and application. He had not been to his office for some weeks past, as he was incapacitated from fixing his attention on those subjects which demanded it, and even to look at a column of figures quite upset him. There was optic neuritis in both eyes, with slight extravasation of blood, turgid veins. and contracted arteries; he could not read No. 2 Jaeger. No other cranial nerves were affected. He had lost power in the left side of the body; his left hand was nearly useless, and showed a grasping power of 25 on the dynamometer, while the right showed 120. The left leg was very feeble, and the left foot apt to drag on the ground, so that the patient avoided walking as much as possible; but there was no reeling gait, such as is seen in cerebellar disease. There was no atrophy in the paretic muscles, which responded equally well to faradisation and galvanisation as those of the right side. Sensation was not impaired, the reflex action of the skin and the mucous membranes normal, that of the tendons appeared somewhat increased. The urine contained an excess of phosphates, but no sugar, albumen, or excess of urea. The action of the bladder and rectum was normal, and there was no sign of disease of any of the other viscera.

Tumour in the right cerebral hemisphere was diagnosed, probably about the ganglionic masses of the corpus striatum and thalamus opticus. The patient was severely questioned about any syphilitic antecedents, but denied ever having had any primary or secondary disease. He had always been extremely steady in his habits, and had never had connexion before marriage. His wife had never had any miscarriages, and she as well as the children appeared perfectly healthy.

I nevertheless instituted an anti-syphilitic treatment, as this appeared to me the only chance of benefiting the patient. One-sixteenth grain of perchloride of mercury was given together with ten grains of iodide of potassium twice a day. The effect of this medication was almost magical. The vomiting ceased after the first dose, and never reappeared at any subsequent time. The appetite began to revive, the headache became less intense, and there was a gradual and steady amelioration in all the symptoms from which the patient had suffered. About a fortnight after commencing the treatment he returned to business, in which he took renewed interest; and rather more than three months afterwards he appeared quite well. No other medicine had been given except cod-liver oil, of which the patient took two tablespoonfuls daily from the third week, and which was continued altogether for six months. The perchloride of mercury had to be occasionally withdrawn for some days on account of affecting the gums too much; but it was always noticed that there was not nearly the same improvement in the more important symptoms when potassic iodide alone was given; and that the patient progressed much more quickly when the perchloride was resumed.

I now proceed to give the details of some cases in which localised lesions of certain portions of the brain, other than tumour, were diagnosed, and in which the catalytic effects of the constant current proved of service.

Case 2. *Diabetes insipidus, Galvanisation of the Medulla.*—A gentleman, aged 37, single, consulted me on July 27, 1880. He had spent many years in the tropics, and had suffered from persistent diarrhœa, which nothing would arrest. This ultimately brought on a state of complete cerebral exhaustion, and he became quite unable to attend to his occupations. The most troublesome amongst the numerous symptoms from which he suffered, however, was that of polyuria, which was so bad as to exclude him altogether from society. When in company, he could hardly sit still for a quarter of an hour without experiencing a most pressing desire to empty his bladder; and this annoyance had led him to adopt a solitary mode of life. The average quantity of urine which he passed

during the day amounted to ninety ounces, but often much more. It was feebly acid, had a low specific gravity, and contained nothing abnormal. As he had taken gallons of physic without the slightest relief, he wished to ascertain from me whether any mode of applying galvanism might be expected to be of service to him.

I have elsewhere[1] given my reasons for considering diabetes insipidus an affection of the medulla, possibly in connection with the middle lobe of the cerebellum, and for looking upon any ultimate organic lesions in the kidneys as secondary, and owing to the maceration of renal tissue through excessive and long-continued diuresis. Proceeding from this view, I directed the current of fifteen cells of Becker-Muirhead's battery to the back of the head, taking care that the medulla received alternately the influence of the anode and cathode, and regulating the finer degrees of voltaic power by the rheostat. The application lasted altogether six minutes, and was entirely painless.

The patient came to see me again a week afterwards, and informed me that the result had been completely successful. The quantity of urine passed during the day had, after the application, fallen to thirty ounces; and while formerly he had often had had incessant calls to pass his water, he had since then only been obliged to pass it three times a day. I regret to say that I had not the opportunity of examining the urine more carefully, either before or after the application of electricity, and that I have since lost sight of the patient, so that I do not know whether the relief has continued. That the change which occurred so suddenly was really owing to the use of the galvanism seems obvious, as no medicine had been given, and the patient had not adopted any alteration in his diet or general regimen. The derangement of the medulla, and possibly of the middle lobe of the cerebellum, which gave rise to the polyuria, must have been slight, and may have been anæmia, or a degree of passive hyperæmia. It was, however, sufficient to render the patient thoroughly miserable, and might perhaps in course of time have developed into more serious organic changes in the organ.

[1] 'Medical Times and Gazette,' November 27, 1880.

Case 3. *Melancholia; Galvanisation of the Occipital Lobes.*—A married woman, aged 28, had her first confinement in July 1877. She had until that time been habitually in the enjoyment of excellent health, and been bright and cheerful in her manner; but soon afterwards a complete change in her disposition was observable. She took a dislike to her husband, seemed to lose all interest in the affairs of her home, and became sullen and morose. She refused to go out, and would sit all day long in a corner of her room, doing nothing. If she conversed at all, it was on religious subjects, in which she had previously shown a very moderate interest only; and the only book she would now read was the Bible. She had a settled conviction that she could not be saved, and was doomed to go to hell. When her friends attempted to reason with her, she became only more rigidly fixed in her ideas, and appeared to resent every interference. I first saw her in March 1878. She had then the expression of a settled frown on her face; the pupils were large; the tongue furred; speech slow and measured. I had much difficulty in inducing her to answer my questions, which she evidently thought impertinent. The appetite was poor, the bowels confined, the catamenia regular. She had never nursed her child. Phosphorus, strychnia, and other nerve-tonics were given, but did not afford any relief. She had been taken away from her home for a complete change, but this had done no good at all. After two months of ineffectual medical treatment and regimen, I proposed the application of the constant current. This was directed to the occipital lobes, with voltaic alternatives, for five minutes at a time. An improvement soon became manifest, and when the treatment was discontinued after twenty applications, the whole aspect of the case appeared altered. The patient was still more reserved and silent than she had originally been, but she was cheerful, and took more interest in the affairs of daily life. She felt more inclined to take exercise, her appetite had improved, and her ideas on religious subjects had undergone great modifications.

Case 4. *Auditory delusions; Galvanisation of the Temporo-sphenoidal Convolutions.*—A gentleman, aged 38, had for some

years past suffered from epileptic seizures, for which he consulted me in February 1879. His memory had become impaired, but his intellect and judgment were good. One morning he came to me in great apparent distress, expressing an apprehension that he was going to become insane, as for some days past he had been subject to delusions, which had come on without any perceptible cause. He constantly heard voices behind his back, calling him names and speaking about his pecuniary affairs and his state of health; and he appeared quite worn out by anxiety on this account. Looking upon this symptom as denoting hyperæsthesia of Ferrier's auditory centres in the superior temporo-sphenoidal convolutions, I applied the current to those portions of the skull corresponding to these parts, for five minutes consecutively. This gave immediate and thorough relief, as the delusion had completely vanished at the end of the application, and did not return on any future occasion.

Case 5. *Corpus Striatum Hæmorrhage.*—A widow, aged 60, had an attack of apoplexy on January 30, 1879. The first symptom was giddiness, which passed within half an hour into complete insensibility. There was involuntary evacuation of the urine and fæces. The patient remained unconscious for about three hours, after which she gradually came to, and appeared next day to be almost herself again, but that there was hemiplegia of the left side. I first saw her ten days after the attack, and found all the symptoms of recent corpus striatum hæmorrhage. There was paralysis of the lower branches of the portio dura, which became plainly visible on laughing and on attempting to show her teeth; she had difficulty in swallowing solids; the tongue appeared bluish, congested and deviated to the paralysed side. Articulation was somewhat indistinct, but the patient had no difficulty in expressing herself. There was no headache or giddiness, but some tenderness on percussion over the right hemisphere. There was no conjugate deviation of the head and eyes. The left arm was completely paralysed, and appeared a piece of inanimate matter, not the slightest voluntary movement being possible in any part of it. The leg, which had at first been as much paralysed as the arm, had regained a slight degree of

motive power, more especially in the knee-joint and the toes. There was also a degree of paralysis in the muscles of the body, for the patient was unable to shift her position in bed, nor could she raise herself from the lying to the sitting posture without assistance. Sensation was normal everywhere; reflex action of the skin and tendons not increased; no ankle-clonus. The faradic excitability of the paralysed muscles was normal. Respiration on both sides of the chest tolerably equal; pulse 90, full, firm; temperature 98·5°; no acute bed-sore anywhere. The appetite was tolerably good, and urinary and alvine secretion satisfactory; she slept pretty well, and was in good spirits. I prescribed a phosphorus perle to be taken twice a day, and recommended the application of the continuous current as soon as ankle-clonus should become manifest. This was first noticed on the twenty-seventh day of the disease. The current was now applied to the right hemisphere, the cathode and anode being alternately directed to the crown of the head and occiput, after which the stream was sent transversely through the brain, from one mastoid process to another. Ten cells of Weiss's Leclanché were used, for eight minutes altogether. This was done on alternate days four times, after which the paresis of the muscles of the body had completely disappeared, the patient being able to sit up in bed and change her position without any trouble. The mobility of the leg had also improved, but there was now slight rigidity in the hamstring muscles. The shoulder appeared more movable, but some rigidity in the biceps and flexor communis digitorum was noticed. The galvanic application to the hemisphere was repeated, and a gentle application to the limbs combined with it. After another week of this treatment the patient had almost entirely recovered the use of the leg, and was able not only to walk about in her room, but also to go up and downstairs without assistance. The rigidity in the muscles of the arm had meanwhile become more marked, but there was also greater mobility, not only in the shoulder but also in the elbow and fingers, while the wrist remained quite motionless. The treatment was pursued with some interruptions for four months. The patient was then able to walk three or four miles at a time without the least

difficulty; indeed all traces of paralysis, as well as rigidity in the lower extremity had disappeared. The arm and hand had not made quite the same progress, but the patient was able to use the hand for dressing and eating; and, although several muscles were still paretic and stiff, she was not nearly so dependent upon others as she had expected to be.

Improvement may follow the systematic use of the voltaic current to the suffering hemisphere even years after the occurrence of the stroke, and when we should expect that secondary sclerosis of the parts below the lesion would render all therapeutical efforts futile. This is shown by the following case:—

CASE 6. *Softening of the Left Hemisphere from Embolism of the Middle Cerebral Artery.*—A single lady, aged 40, had had a stroke which took away her speech and the use of the right side of the body, in summer 1868. I first saw her in July 1878, that is, ten years after the occurrence of the attack. The early history of the case could not be very fully ascertained, but there was little doubt from the symptoms present that she had suffered from embolism of the left Sylvian artery, with consecutive necro-biotic softening in the sphere of that vessel. There was now distinct aphasia, the patient being only able to say "yes," "no," and a few other words, such as "doctor," "my hand," &c.; yet she understood everything that was said to her, could read the paper, and express her wants by pantomime. She behaved quite rationally, and her intellect seemed to be good. There was slight affection of the lower facial muscles; the tongue was thin, and could only be protruded a very little way, but was not deviated. The whole right arm was in a state of paralytic rigidity, and utterly useless. The wrist could neither be raised nor pronated nor supinated, and the fingers were so powerfully flexed that the nails touched the skin. The hamstring muscles of the thigh were likewise contracted, and the patient, although able to walk, was very lame. There was no wasting of muscles, which responded well to faradisation. Ankle-clonus was well marked. The general health of the patient was good, except that she had three years ago lost the catamenia, since when she suffered from periodical headaches.

A similar treatment was resorted to in this case as in the

above, and continued for six months. At the end of that time the rigidity of the leg had almost entirely disappeared, and the patient walked with great ease. There was considerable amelioration in the use of the right hand, which could be used for dressing, eating, needlework, embroidery, etc., but not for writing, which the patient had learnt to do with the left hand. The most satisfactory feature, however, was the improvement in her language. The patient had regained the command of a large number of words which she had lost before; she was able to read aloud and to converse, and on many occasions pronounced long sentences with tolerable fluency. She was however easily flurried, more especially in the presence of strangers, and then would begin to stammer and to lose the thread of the conversation; she would, however, recover herself after a few minutes, and then speak comparatively well again. The catamenia reappeared after the galvanic treatment had been followed for some weeks; and, although they did not again become thoroughly regular, they continued at intervals varying from six to ten weeks; and she lost the periodical headaches to which she had been subject before.

The precise mode of action of the constant current in destructive brain-lesions will probably not be understood until a number of cases which have been treated in a similar manner have been thoroughly investigated post-mortem. Is there true regeneration of nerve-fibres in the internal capsule under such circumstances, and can secondary sclerosis below the lesion be repaired when the conduction between the cortex and the periphery becomes, at least to some extent, re-established? That repair of destroyed nerve-fibres may occur has been shown not only by experiments on animals, but also by cases of neurectomy for trigeminal neuralgia, in which anæsthesia owing to loss of nerve-substance (amounting in some cases to as much as three-quarters of an inch) was recovered from, and the paroxysmal pain, which had disappeared after the operation, returned in the same sphere after a time. This points in the most unmistakable manner to re-established conduction. Can part of the internal capsule be repaired in a similar manner? Or are the therapeutical results of galvanic applications owing to a more complete removal of the causes

of inhibition, and to the induction of vicarious function on the part of neighbouring structures? These questions have a great theoretical as well as practical interest, and demand the serious attention of neurologists. In the meantime not too much stress can be laid upon the importance of treating such lesions at an early stage, and before secondary sclerosis has been fully established. With regard to this point the occurrence of ankle-clonus after an apoplectic or paralytic seizure should be carefully watched for, as it is a diagnostic sign of considerable value leading us to predict the probably speedy appearance of late rigidity in the paralysed muscles; and as soon as this symptom has made its appearance, no time should be lost in resorting to the use of electricity.

OBSERVATIONS ON CERTAIN OPTICAL ILLUSIONS OF MOTION.

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It is proposed in the present paper to consider some of the arguments and conclusions recently advanced by Professor Silvanus Thompson upon this subject,[1] and to offer additional suggestions. In order as far as possible to avoid repetition, the present writer will assume that his readers are familiar with the contents of Professor Thompson's article. By the courtesy of that gentleman, the figures which illustrated his paper are here reproduced for convenient reference.

First, as to the *law of subjective complementary motion*, which Professor Thompson enunciates. It appears to afford a satisfactory explanation—the only one which can at present be offered—of certain particular optical illusions of motion not explicable by any other theory. The new law is as follows:—"The retina ceases to perceive as a motion a steady succession of images that pass over a particular region for a sufficient time to induce fatigue; and on a portion of the retina so affected, the image of a body not in motion appears by contrast to be moving in a complementary direction."[2] By this law the following illusions are capable of explanation: 1. After looking from a rapidly moving railway-carriage at objects lying beside the line, as they pass before the eyes, let the eyes be closed suddenly, an apparent motion in the opposite sense will at once be perceived, undistinguishable forms and patches of light seeming to rush past the blank field. 2. After looking for some time at a waterfall, and then at the rocks adjacent, the rocky surface may be seen to appear as if in motion upwards. 3. The appearance of reversed rotation which

[1] 'BRAIN,' Part XI., October, 1880. [2] L. c., Vol. III. page 296.

is seen after looking for some time at a slowly revolving disc divided into black and white sectors (Fig. 3). 4. The apparent expansion of surrounding objects after looking at the end of a retreating railway-carriage. 5. The apparent convergence to, and divergence from, a given centre, of objects looked at after the eye has been fatigued by regarding a revolving disc of black and white spirals (Fig. 4). As regards these illusions and others of a similar nature, the "muscular slipping" theory fails entirely, but Professor Thompson's new law is perfectly satisfactory.

The apparent rotation of the concentric circles (Fig. 1)

Fig. 1.

cannot, however, be explained by this law. Professor Thompson himself says that "the persistence of visual impressions has a good deal to do with the production of this illusion," but he does not explain in what way. The following appears to be the true explanation of the manner in which the persistence of visual impressions gives rise to the appearance of rotation of "strobic circles" to which a "rinsing" motion is imparted; and it is believed that by it all the facts of this curious illusion can be explained.

If the circles be "rotated" as slowly as possible, it may

be noticed that the appearance of rotation is due to the seeming revolution of apparent radii or sectors. The whole disc appears divided into four sectors—two narrow ones, which are opposite to, and in a line with, one another, and in which the black and white bands are distinctly defined—and two much wider ones, in which the black and white rings become somewhat lost in one another, causing a blurred appearance. These sectors may be best seen by moving the circles somewhat rapidly upwards and downwards, that is, vertically, without lateral motion. The clearly-defined, narrow sectors will then be seen extending horizontally on either side of the centre, while the upper and lower portions of the disc appear somewhat indistinct, brownish, and blurred. If the disc be oscillated laterally, without vertical motion, the clear sectors at once assume a vertical direction; they are, in fact, always perpendicular to the direction of motion.

The explanation of this appearance is obviously to be sought in the persistence of retinal impressions. During vertical motion, the images of the more or less horizontal black and white bands which occupy the upper and lower parts of the disc are constantly replacing one another upon the retina, each becoming confused with the impression immediately preceding it, and thus producing the blurred appearance noticed. The reverse is the case in the narrow lateral sectors, in which the black and white bands are practically vertical, and coincide with the direction of motion; their images consequently do not displace one another upon the retina, but remain clearly defined and unaffected by the vertical movements. It is very difficult to produce, with the unaided hand, to-and-fro movements which shall be either purely vertical or purely horizontal; the result of this is that, in trying this experiment, the clear sectors do not remain absolutely fixed in position, but oscillate in the sense of the imperfections of movement, reminding one much of the behaviour of the magnetic needle when shaken or jolted.

In the rinsing movement, which produces apparent rotation of the circles, the disc is moved in all directions successively during each revolution: the clear sectors being, as we have seen, always perpendicular to the direction of motion, must,

therefore, also move in all directions successively, following the direction of motion, though constantly at right-angles to it. An appearance of revolving radii, similar to that produced by the spokes of a rapidly revolving wheel, is thus brought about. It is almost needless to point out that if a series of perfectly concentric black and white circles were set revolving, no such appearance as that now described (of revolving radii or sectors) would be produced; indeed, if the bands were of unvarying width, and with very clearly defined borders, it might be difficult to discern, apart from other evidence, whether revolution were really taking place or not.

It will be found that several other curious facts connected with this illusion are easily explicable in accordance with the explanation just offered. The reason why the circles appear to revolve with the same angular velocity as that imparted is at once obvious.

"It is found that for increasing distances from the eye the concentric rings must be made wider if the illusion is to succeed; there being apparently one particular magnitude of their images on the retinæ, which favours the production of the illusion."[1] This quite accords with the explanation offered; the black and white bands must be large enough to be easily and clearly distinguishable by the eye when they are in the clear sectors of the disc; but they must not be so large as to interfere with the necessary confusion of the images received upon the retina from the greater part of the disc's surface. If the bands were of a certain magnitude, it would be competent for the eye to fix itself on one particular band and follow its movements in every direction, in that case there would be no rapid sequence of images passing over any portion of retinal surface, and no illusion would be produced. The happy medium in the size of the bands, which is necessary to the success of the illusion, must obviously, in accordance with this explanation, vary with their distance from the eye.

If two sets of circles printed upon one card be "rotated," those at which the eye is not looking revolve most vigorously. This is another way of saying that the circles rotate best when their

[1] L. c. p. 293.

images fall upon parts of the retina which are somewhat removed from its centre. The circles always rotate best if the eye be fixed on some point a little outside them. My explanation of this fact is that, the more peripheral parts of the retina being less sensitive than the central, the psychological element plays a larger part in the interpretation of the impressions received, and, being supplied with more imperfect data, is more liable to arrive at erroneous conclusions as to the mode of causation of the images perceived. The physical impressions being defective, what wonder if the psychic faculties fail to attribute them to their true origin?

Again, "on stopping the 'rinsing motion' suddenly, there appears to be, for an instant, a reverse motion." [1] For this, I believe, there are two causes; the first and most important is to be found in Professor Thompson's law of subjective complementary motion. The second cause is that the hand, in trying suddenly to stop the circular movements, often involuntarily commences a revolution in the opposite direction, thus causing the clear sectors also to reverse the direction of their movements for an instant.

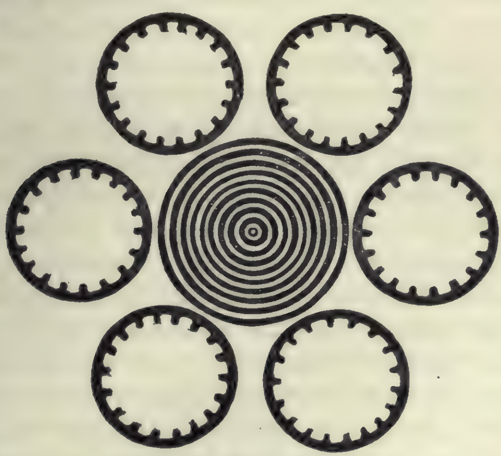

Fig. 2.

Coming now to the toothed wheels (Fig. 2), I believe that their apparent rotation during "rinsing movements" must

[1] L. c. p. 293.

be explained by yet another theory. The mere fact that the wheels only move through one tooth while the circles make a complete revolution, and in a contrary direction, is of itself sufficient to indicate that the two illusions are essentially distinct and susceptible of different explanations. It seems to me that it is here that the "muscular slipping" theory, or something very like it, comes in. It is evident that the eye, from some cause or other, once in each revolution, mistakes the image of each tooth for that of the one next adjacent; the wheel thus appears to have moved onwards through one tooth.

Adopting the "muscular slipping" theory, the following would be the sequence of events. The eye follows the movement of each tooth for a certain distance, but, not moving fast enough, it catches sight of, and slips back to, the next following tooth, and mistakes it for the original one. Of course, the persistence upon the retina of the original impression aids materially in bringing about the apparent coincidence of the two images.

The matter may perhaps be put more correctly thus: the eye, in order to avoid the oscillations necessary to follow each movement of the teeth, remains almost stationary, and passively accepts the image of the next tooth which appears near the position of the previous one, as a continuance of the original impression, which original impression has, in fact, persisted until, or nearly until, the new one takes its place.

The toothed wheels rotate far better when the eye is not directly fixed upon them; in fact, this is a matter of much greater importance with the toothed wheels than with the strobic circles. This explains why the wheels are so effective when arranged round concentric circles, as these latter divert the eye from regarding the teeth too minutely. It is possible, with some effort, to fix the attention for a time upon one single tooth, in such a way that the illusion cannot be caused by rinsing movements, unless these be more rapid than can be produced by hand.

It will be found that the disc of black and white spirals, shown in Fig. 4, may be made to "rotate" perfectly if the eye be kept fixed upon the centre of Fig. 3, while rinsing

movements, with a somewhat large radius of motion, are made. The great indistinctness of the images formed upon the peripheral parts of the retina, together with their persistence,

FIG. 3.

FIG. 4.

allows of each black spiral being in turn mistaken for the one next to it. In accordance with this view, the disc should rotate once for each three revolutions of imparted motion. This is apparently the case, but it is difficult to count the revolutions, as the illusion ceases immediately the eye is fixed upon the figure for that purpose. If the eye be kept fixed upon one of the spirals, the illusion cannot be produced; unless, indeed, the eye has become considerably fatigued.

Three distinct causes of optical illusions have now been noticed. First, that given in Professor Thompson's new law; little has been said concerning it, because it is fully explained in the paper in which it is enunciated. The other two, represented respectively by the illusions of the strobic circles and the toothed wheels, have been more fully considered. The correctness of the views here given, as concerning the particular phenomena attempted to be explained by them, in no way implies that optical illusions of motion are not produced in various other ways. I mention this because Professor Thompson rather appears, in his paper, to proceed upon the assumption that, because the muscular slipping theory can be proved not to apply in certain cases, it therefore does not apply in any.

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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(Continued from Vol. III., p. 515.)

III.

Œdematous Conditions of the Brain.

An œdematous state of the cerebral tissue accompanies all cases of softening of the brain, whether of inflammatory or non-inflammatory origin; but it is thought desirable to give an independent consideration to cerebral œdema, often the only morbid condition recognised by the naked eye in the brain of the chronically insane. As it is chiefly compensatory in its origin, it will be considered together with that compensatory effusion into the ventricles and beneath the membranes of the brain which so frequently accompanies it. If the cerebral tissue becomes much infiltrated with serum, it is liable to break down the texture of the brain. As before stated, this may proceed to a state of white softening, in which the brain tissue may become quite diffluent, such as we observe in the neighbourhood of the lateral ventricles in acute hydrocephalus and around inflammatory foci. The more frequent condition met with in the brain of the insane is that of a general moist condition of the white matter, which is soft, almost pasty, clings to the blade, and is apt to tear away in shreds of a dirty white hue. Such is the appearance in cases of senile wasting of the brain. The white medullary strands immediately bordering upon the lateral ventricles will usually be found most implicated, being here in closer contact with the serum contained in these cavities. The lesser

degrees of œdema are recognised by pitting of the tissue on pressure and a peculiar and notable brilliancy of the white substance when cut across, one of the most frequent appearances in the brain of the insane. The student must learn to recognise the *general moisture*, *swollen condition*, alterations in *consistence*, and alterations in appearance from a *brilliant to a dull dirty-white hue*, associated with varying degrees of œdema. If the freezing microtome be liberally used, he cannot fail to appreciate to the fullest extent the varying degrees of œdema of the cerebral tissue; and this not only because it proves the most troublesome obstacle to a free manipulation of delicate sections, but from the tendency of œdematous brain to freeze into a hard icy solid, differing much from the consistence of healthy brain when frozen. It is impossible to cut through such frozen brains except by a modified process to be alluded to further on. In the hardening processes, also, these brains undergo by dehydration enormous shrinking. The serous effusions beneath the arachnoid and into the ventricles, which have been alluded to as often associated with œdematous conditions of the brain, may occur as the result of—

a. Senile atrophy of the brain (compensatory).

b. Pressure by morbid products on the vessels, as in—
  - Tubercular meningitis.
  - Syphilitic disease of blood-vessels.
  - Tumours.
  - Abscesses of mid-lobe of cerebellum pressing on the venæ Galeni.

c. As a gradual accumulation from anæmia and other existing morbid states, as in chronic phthisis.

Where does the effusion of serum attending œdema of the brain occur?

a. Into sac of arachnoid, i. e., betwixt the cerebral layer of the arachnoid and the polished inner surface of the dura mater. On removal of the brain, the student will note this fact by examining the amount accumulated in the occipital fossæ above and below the tentorium.

b. Beneath the cerebral arachnoid. Observe how the latter is floated up by the subjacent fluid where this membrane bridges across the sulci.

c. In the meshes of the pia mater. Note its swollen gelatinous appearance from infiltration with serum.

d. Into the ventricles :—

1. More or less distending the lateral ventricles.
2. Stretching tense the thin arachnoid which extends between the cerebellum and medulla over the fourth ventricle.

Before discussing the subject of œdematous conditions of brain and compensatory effusions of serum, we would recommend the student to acquire information upon the following facts in all such cases :—

1. Condition of ependyma (lining membrane of ventricles),
   a. As to healthy aspect. *b.* Granular appearance and feel. *c.* Macerated aspect. *d.* Fibro-cartilaginous plates.
2. Turbidity or discoloration of serous fluid.
3. Specific gravity of serous fluid.
4. Reaction to litmus paper.
5. Specific gravity of the brain.

Augmented Consistence.—All cases of induration of the brain should induce the student to examine the morbid change in the texture as regards its *degree*, its *distribution*, and *nature*. The cases most likely to present themselves to his notice may be embraced under the following categories :—

1st. General augmented firmness of the whole brain from increase of its connective or neuroglia element.

2nd. Limited but extremely indurated patches or nodules due to morbid growths, especially the carcinomata.

3rd. Limited indurations of grey or white matter due to transformation of inflammatory products.

4th. Sclerosis disseminated throughout the brain or distributed down the motor strands.

The student will observe that the increase in consistence may vary from a scarcely appreciable degree, being due merely to a lessened quantity of water (Rokitansky) up to a condition of leathery callous consistence.

A few words upon the varieties of sclerosis met with in cerebro-spinal centres. First the student will become familiar with the general increased firmness of the brain from in-

crease of its neuroglia on examining the brain of many epileptics.

Superficial Scleroses.—He should also look for instances of *partial sclerosis of the grey cortex* occasionally to be seen in epileptics and imbeciles, and characterised by a peculiar shallow puckering of the surface of the gyri, which are somewhat indurated and have a cauliflower appearance.

Hypertrophy of Neuroglia.—Again he may meet with instances of hypertrophy of the brain, as it is termed, and which has been found due to extreme increase of the neuroglia around the medullary strands of the hemispheres. Such a case would present him with increase in *consistence, elasticity,* and *volume.* In all cases of general increased consistence it is imperative that the greatest attention should be paid to examination of the brain under the following heads :—

a. Volumetric measurement.

b. Absolute weight of the brain and its divisions.

c. Specific gravity of grey and white matter.

Cicatricial Formations.—The indurations resulting from inflammatory action will frequently attract attention. Such patches of callous tissue may surround foci of softening of the cortex due to plugging, or the site of old hæmorrhages. Cyst-like cavities, with hard fibroid walls, may thus be formed deep in the substance of the hemispheres. Sections through such structures should be made for microscopic examination, as they illustrate well the various stages of transformation of inflammatory products into callous cicatricial tissue.

Disseminated Sclerosis.—If in slicing a brain greyish nodules are found scattered irregularly through the white substance resembling the cineritious substance in colour, but becoming of a rosy hue on exposure and exuding a colourless fluid, the student has probably to deal with a case of disseminated sclerosis. Note in such a case the increased consistence of the sclerosed patches, their vascularity, irregular distribution through the medullary substance, and the comparative immunity from this lesion enjoyed by the cortex. Sections should be preserved for microscopic examination. The extent of implication of the ganglia, pons, medulla, and spinal cord should also be examined.

Descending Sclerosis.—If again a greyish induration should be met with involving the white strands running down from the motor region of the brain to the internal capsule, or from the latter down into the crura, and on into the lateral columns of the cord, we are dealing with a secondary descending sclerosis (fasciculated sclerosis of Charcot).

B. *Colour.*—The alternating lighter and darker shadings of the cortical layers vary much in the depth of their hue, rendering the distinctness or differentiation of the individual layers more or less apparent. The variations in hue from the normal standard may be distributed in *mottled patches or laminated zones*, or *spread uniformly throughout the whole cortical envelope.* The student will do well to consider the conditions usually associated with alterations in the colour of the cortical layers, and first and most frequent amongst such conditions must be placed *alterations in the blood supply.* The next most frequent condition met with is *disintegration of nervous tissue;* and next to these in their relative order of occurrence we find *morbid deposits;* and lastly, *new growths* in the cortex.

Let us first consider the morbid conditions associated with unusual pallor.

a. Partial Mottling and Laminated Pallor.—A very slight acquaintance with the morbid appearances found in the brain of the insane will suffice to attract attention to the frequent presence of irregular, oval or circular patches of pallor, very limited in extent, and of a yellowish grey hue. These patches may extend throughout the whole depth of the grey matter or be limited to the superficial layers alone. Now this condition appears almost invariably attributable to anæmic zones, in which special systems of arterioles are involved. It may however be due to granular disintegration of nerve-cells and fatty accumulations in the perivascular sheaths.

A similar patchy pallor will also be frequently met with in the central ganglia, but here it will usually be found associated with fatty degeneration of the nuclei in the walls of the blood-vessels.

A portion of a convolution exhibiting this appearance should be placed on the freezing microtome, with the blotchy

aspect uppermost. On freezing it will be observed that the whole depth of cortex assumes a uniform pallor throughout, and sections cut off and floated in water *fail to exhibit the pale patches* if the latter be due to anæmic conditions simply; on the other hand, if due to structural disintegration, the appearance vanishes during the frozen state, only to return rapidly when the section is cut. The process of hardening by chrome salts dissipates all patches of pallor due to anæmic zones, for by both processes the blood vessels are more or less emptied of their contents, and a uniform tint results.

Apart from the presence of fatty or granular débris, the general pallor of the cortex may be due to great paucity of nerve-cells, whilst the blood supply being diminished the resulting pallor will be associated with a poor differentiation of layers. This apparent fusion of layers and loss of the sharp boundary lines seen in healthy brain is therefore a fact of some significance; it almost invariably points to general malnutrition and disintegration of nerve-cells. Pallor, however, is not necessarily associated with disintegration of nerve-cells, for the latter condition is frequently attended by vascular injection.

b. General Diffused Pallor.—I cannot do better here than describe to the student the appearances found in the case of a patient exhausted by phthisis, in which the typical *pallor* and *malnutrition* of the nervous centres is always so prominent a feature. Such cases are unfortunately so frequent in our asylums that the student will have little difficulty in procuring such a brain for study.

Upon removal of the extremely thin, blanched skull cap, and reflecting the dura mater, the subjacent membranes were seen separated widely from the surface of the brain, buoyed up by fluid which in part was clear and translucent, in part slightly turbid from films and flakes of lymph. The small arterial branches were not visible, the larger primary branches being alone apparent at the vertex. The venous system was represented by engorged trunks, their minutest radicles being well seen. The arachnoid where it crossed the sulci was more or less cloudy, the milky opalescence being due to interstitial change and lymph deposits. In the postero-parietal regions,

where the membranes were floated by the distending fluid off from the surface of the gyri, the smaller veins could be traced meandering through the fluid, and dipping down to enter the cortex. Tracing the course taken by these vessels, the smallest radicles were seen emerging from the cortex, uniting here and there with larger twigs, which ramify through the serous fluid and cross obliquely or directly over the summit of the convolution, to terminate in the primary veins which are formed by their convergence. These latter veins run up the sulci on either side of the convolution, exposed superficially and directed towards the median line or longitudinal fissure, where they terminate in the longitudinal sinus. To render their distribution more apparent, we have only to compress the orifice of one of the larger superficial veins near the median line, whilst with the handle of the scalpel placed on a distal part of the same vein, we include numerous converging radicles betwixt the occluded points. Upon gradually drawing the handle of the scalpel along the course of the vein upwards, the blood is projected into these radicles, and innumerable branchlets before invisible strike across the field over the convolution, or meander in tortuous arborescent forms through the subjacent fluid. The student will learn from the above examination the following facts:—

1st. The larger arteries being at the base, the smaller branches are alone seen near the vertex, and these usually deeply seated in the sulci.

2nd. All the larger blood-vessels usually exposed at the vertex are veins.

3rd. The direction taken by the minute venous radicles will be accurately learnt.

4th. A high degree of vascularity simulating passive congestion may be induced *artificially* with ease by slight force.

It is necessary to observe here that great caution should be observed by the student in hastily arriving at any conclusion as to the presence of congestion of the brain from a superficial view of the veins at the vertex—upon this point the tyro constantly errs. "We must make it a rule to consider hyperæmia of the cerebral membranes as proved only in those

cases where the finest vessels are also injected, and where the overloading of the cerebral vessels is not at all in proportion to the amount of blood in other organs."[1] So says Niemeyer, reiterating a caution constantly given by pathologists, and as frequently neglected. Proceeding with our study of the anæmic brain, the membranes were next stripped and two facts noted; they were slightly thicker and more tough than in health, and they were removed with extreme ease. The student must be familiar with the difficulty experienced in stripping the membranes from healthy brain, being recommended often to remove them under water. This difficulty is not alone due to the tenuity of the pia mater and arachnoid, but also to the fact that the former membrane is attached to the surface by the prolongations of connective cells recognised as Deiter's corpuscles. These are by no means numerous in health, yet sufficiently so to keep the pia mater in firm contact with the cortex. These cells are chiefly found in the neighbourhood of the vessels, where they enter the cortex from the pia mater, and both together form an impediment to easy stripping. In cases of subarachnoid serous effusion such as the one under consideration, the membranes have been floated up from the receding surface of the atrophic cortex, and the connections with Deiter's corpuscles have therefore been forced asunder. After noting fully the consistence of the brain according to the plan already given, a section was made exposing the centrum semi-ovale, and the following facts presented themselves.

1st. The blanched aspect of the cortex and the absence of any reddish striation of the upper layers such as is seen when the cortical vessels are full of blood. The hue of the cortex was *ashy grey throughout*, except in its most vascular zone, where perhaps the slightest warmth of tint was recognisable, but beyond this nowhere did any blush suggest the presence of its extremely elaborate vascular apparatus.

2nd. The layers were individually very poorly differentiated.

[1] Niemeyer's 'Practical Medicine,' translated by Drs. Humphrey and Hackley, vol. ii. p. 155.

3rd. The medulla was brilliant white, glistening, slightly reduced in consistence, and showed few or no puncta vasculosa.

The student will do well to impress the fact upon his mind that the vessels of the meninges may be tortuous and turgid with blood whilst the minute vascular supply of the cortex is absolutely diminished, and the layers appear blanched and anæmic. To a certain extent this was the case in the brain we have under consideration. How this occurs (passive or active hyperæmia of the membranes associated with anæmia of the cortex) will be explained in our next section, when speaking of hyperæmia of the brain. For the present, let the student mark well the fact that the *vascular condition of the membranes is no index*, as a rule, to the *state of the cerebral substance.*

The brilliant white glistening aspect of the medulla was due, as will be shown further on, to a slight degree of œdema of the brain substance.

Let us now place a small portion of a parietal convolution of thin and anæmic brain upon our freezing microtome and cut a fine section through the cortex—float it on the glass slide and drain off superfluous fluid. Take a hand lens and examine first by reflected light. The cortex is seen apparently to consist of—

1st. An outer light translucent zone, extending half way down.

2nd. A deep opaque white layer extending to the medulla, which is still more opaque and white in aspect.

Raise the slide and examine by transmitted light. We now see—

1st. The narrow translucent grey of the first or outer layer of the cortex.

2nd. A broad less translucent grey zone of the second and third layers.

3rd. A broad stripe of darker grey extending to the medulla, but separated at one-third its depth by a narrow blue belt.

We see thus by the naked eye the constitution of the cortex in this region of the brain, except that the narrow second layer cannot be defined from the third layer by *unaided vision.*

Let us turn to the cortex prior to section cutting and examine by means of a hand lens under reflected light, and the appearances are as follows:—

1st. A grey belt corresponding to the first layer.

2nd. A broad white belt divided midway by a narrow grey line.

3rd. A grey belt constituting the deepest layer.

We have, I think, gone sufficiently into the appearances presented by the anæmic brain for our present purpose. The examination of the various layers above recommended will prepare the student, not only for recognising the relative position of layers which have later on to be minutely studied, but will teach him to estimate roughly—

a. The differentiation of the layers by naked vision.

b. The relative depth of individual layers.

c. The atrophy or normal depth of the cortex.

d. The presence of morbid products.

Of the latter I need here only mention those cases of so-called miliary sclerosis in which the nodules can be distinctly seen in sections on examination by *reflected light* dotting the surface over with minute opalescent spots. For the present we must relegate such cases of morbid formations to the chapter on the microscopic appearance of the morbid brain, and now recapitulate shortly the points to be noted when examining by naked vision a cortex of unusual pallor:—

1st. The extent of pallor throughout the brain.

2nd. Its limitation in depth through the cortex.

3rd. Its disposition in patchy areas or otherwise.

4th. Its association with obscure laminar boundaries.

5th. Its association with pigmentary tints.

6th. Its association with altered consistence and œdema.

7th. Its reappearance in sections from frozen brain.

8th. Examination of sections by transmitted and reflected light.

Redness.—The depth of tint acquired by the grey layers of the cortex depends not alone on the presence of pigmented nerve-cells, but also on the far greater vascularity of the grey as compared with the white matter, whilst the layers most richly supplied with nerve elements possess also the more abundant

capillary supply. The more vascular the layer is, the deeper and warmer will be its tint. Now the capillaries of the cortex are of remarkably fine calibre, and hence, when the ultimate arterioles are injected, the appearance resulting is that of a *uniform rosy blush* more or less dark, the surface *smooth* and *swollen*. This uniform coloration, if at all extreme, is often attended by slight extravasations of blood, and the tissue around presents a diffused staining from blood pigment. Such are the conditions found in genuine congestion of the cerebral substance, and the analogous condition is readily recognisable in the soft membranes. The student must make himself thoroughly acquainted with the evidences of a genuine active or passive congestion of the brain, and learn to distinguish it from actual inflammation where, as already pointed out, the *consistence* as well as *colour* are profoundly implicated and the *specific gravity also affected.*

a. Congested zones.—One of the most frequent appearances in the cortex of the acute forms of insanity is a bright arterial zone which bounds the confines of the white and grey matter of the convolutions, following out accurately the direction of the innermost cortical layer. This congested belt corresponds to the horizontally disposed nexus of blood-vessels into which the larger straight cortical arteries empty themselves after passing through the various cortical layers. Any undue engorgement of the vessels of the pia mater will necessarily affect these larger blood-vessels, and from their general arrangement a mechanical element is introduced which is significant of a safety-valve action for the cortex in cases of vascular engorgement, relieving the cortex somewhat by their distension from congestive conditions. It is on this account, I believe, that this linear vascularity is so often apparent without a corresponding blush in the cortical layers, indicative of distension of the minute capillaries.

b. Limited Patchy Congestion of Cortex.—When, however, the cortex is itself congested the student will frequently observe irregular bright red patches, suggestive, like the anæmic patches already referred to, of the implication of minute vascular areas, whilst leading down to them are seen the distended straight cortical vessels, giving the upper layers

a reddish streaked aspect. The last act of arterial contraction in which the smaller arterioles have failed to empty themselves into the venous system may in part explain this appearance, and caution is necessary lest we hastily assume that this state is the result of morbid activity. The student should therefore examine closely the injected part by the microscope and look for evidence of minute extravasations, hæmatine crystals, and staining as well as broken down texture, or other results of inflammatory action and of congestion. This blotchy red aspect of the cortex reappears very frequently in the medulla in similar cases, and is indicative of engorgement of the minute capillaries of these regions.

c. Puncta Vasculosa a fallacious guide.—It is customary, in judging of the degree of engorgement of the vessels of the white matter, to be guided by the paucity or abundance of the puncta vasculosa caused by section of the engorged channels; in other words, the number of drops of blood oozing from the divided vessels are supposed to form some criterion of the engorged condition of the vascular system here. It has been shown by Niemeyer that this is no safe criterion, as

1st. The number of puncta vasculosa vary greatly with the fluidity of the blood, which is an important element in their causation.

2nd. They are often almost entirely absent, when we have had undisputed evidence of intense vascular engorgement during life.

The student must therefore be on his guard against attributing too much importance to abundant bloody points on section of the medulla, whilst he should never fail to note whether this condition is or is not associated with distension of the minute capillaries, as indicated by fine diffuse patchy redness.

I need not here dwell upon the changes of colour due to inflammatory action, as these have been already disposed of when illustrating anomalies of consistence; but I must here repeat the important caution, that the student be not misled by expecting to find engorgement and redness in all cases of encephalitis. The larger number of such cases present no undue red coloration.

Results of Intra-cranial Pressure.—The student must invariably allow for conditions of increased intra-cranial (*but extra-vascular*) pressure. When the cranial cavity is encroached upon by a tumour, abscess, hæmorrhage or serous accumulation, compensation is made by the outflow of the subarachnoid fluid into the spinal meninges. The *limit of this compensatory arrangement* is soon reached, and then the only available space is that gained at the expense of the *general vascular calibre.* Encroachments upon the cranial cavity therefore eventually empty the blood-vessels, causing partial or more or less general anæmia prior to compression of the brain substance itself. It is therefore far from improbable that, as Niemeyer teaches, engorgement of the blood-vessels may reach such a degree that after the limit of subarachnoid compensation has been reached, effusion of serum occurs into the perivascular spaces and brain tissue, and suffices to compress the minute blood-vessels and capillaries. Hence we may find, in such cases, an association of marked cerebral anæmia with tortuous and engorged blood-vessels in the meninges.

Results of Intra-thoracic Pressure.—Venous engorgement of the brain is a frequent result of obstructed circulation through the medium of pleuritic effusions, intra-thoracic growths, fibroid induration and other changes obstructing or obliterating the vascular system of the lungs. Occasionally, though rarely, there is found an extraordinary engorgement and varicosity of the veins of the pia mater—the vessels winding in all directions and, as Rokitansky states,[1] even in spirally twisted coils and intestine-like circumvolutions. In these cases the brain substance will be found dark and engorged, full of puncta vasculosa and frequent miliary apoplexies. In one very typical case, occurring at West Riding Asylum, the brain surface was actually concealed over extensive tracts by a vast development of varices and contorted vessels, and when cut into, the grey and white substance was not only deeply engorged but presented dozens of miliary hæmorrhages forming patches varying from a pea to a florin in extent, and in all

[1] 'Pathological Anatomy,' Sydenham Soc., vol. iv. pp. 372–3.

degrees from the purely punctiform to the diffuse uniform extravasation. The appearances of a section of this brain are well shown in a preparation at the Asylum Museum. A similar case was exhibited some years back by Dr. Coupland at the Pathological Society of London.[1] In his case the mechanical hyperæmia was due to acute bronchitis; in the Asylum case, the immediate cause was the supervention of capillary bronchitis upon fibroid lungs in a patient who gave a history of intemperance. Its association with drink has been recognised by Rokitansky. The occurrence, then, of such cases should direct the student's attention to the effects of intra-thoracic pressure upon the venous circulation of the brain.

Volumetric Methods.

Cubic Measurement of Brain.—The bulk or volume of the encephalon can be very readily ascertained, and, considering the important information it affords, it is a process too frequently neglected in our post-mortem rooms. The volume of the brain is estimated by its displacement of fluid. Dr. Hack Tuke has detailed, in the January number of the 'British and Foreign Medical Review' for 1855, the result of examination by this method of sixty-three brains, together with the capacity of the crania. The vessel used by this observer was one of convenient size and shape, with a capacious spout placed at an acute angle with the sides. Water is poured into this vessel up to the level of the spout. Fluid contained within the ventricles and subarachnoid space is allowed first to escape by several long incisions, and then the brain, including the medulla oblongata, is immersed—the displaced water, as it escapes from the spout, being caught and measured, affording an exact criterion of the actual bulk of the brain. The student may, however, prefer the use of a graduated vessel upon which he can at once read off the displacement of fluid, but the exact graduation of large vessels such as would be required is open to several fallacies; and these possible sources of error are, of course, wholly avoided by Dr. Hack Tuke's method. If such a vessel as that used by Dr. Tuke be not at

[1] Reported in the 'Lancet' for January 11, 1879.

hand, a ready method is the one I have used, as follows:—half fill with water an inverted bell glass of sufficient dimensions, and mark the level of the fluid. Now immerse the brain in the fluid, and note the level of displacement, after which the brain is removed; if the original level is not exactly maintained owing to imbibition by the brain, pour in sufficient fluid to compensate for the loss. Water is now poured in from a graduated measure up to the displacement level, the amount required for this purpose giving us the volume of the brain. The volume should be estimated in cubic centimeters and cubic inches.

When describing the methods of section-cutting I shall refer to a vessel which will answer well for the volumetric estimate of the brain. It consists simply of a large glass vessel such as is used for the preservation of brain in museums, but fitted with a stop-cock arrangement in the side near the bottom of the vessel. It is filled with sufficient water to cover the brain, the level being marked or indicated by a weighted float. The brain is next immersed, and whilst so immersed, the fluid of displacement is run off by the stop-cock, and measured in a graduated vessel. Again, in lieu of a graduated vessel, the student may employ a float, moveable along a graduated weighted stem (graduated, of course, for the vessel used). Such a float should terminate below in a brass button, which rests against the bottom of the vessel, keeping the graduated stem in a vertical position, whilst the float freely moves with the rising or falling level of the fluid along the graduated stem.

When referring to the specific gravity of the brain, an apparatus will be described whereby volume as well as weight of brain are readily measured. Such an apparatus has the recommendation of being cheap and efficient. Whatever arrangement be adopted, we should not rest content with the estimate of the volume of the encephalon alone. Each hemisphere should be separately measured, as also the cerebellum, pons, and medulla. It will be found convenient to use smaller and more delicately graduated vessels for the latter.

Cranial Capacity.—This should be estimated with a view to

a comparison between the actual volume and weight of the brain and the dimensions of the cranial cavity, as well as for comparison with the capacity of average-sized skulls. The relationship between cranial capacity and brain weight has been shewn by Dr. Barnard Davis, who has adopted the general rule that a deduction of about fifteen per cent. from the capacity of the cranium gives the "capacity" of the brain, whereby its weight may be readily calculated.[1] The large bulk of cranio-metric observations having been taken upon the dried skull, we should when dealing with the fresh subject make allowances for slight divergence; and, in order to approximate the conditions, the dura mater should be wholly removed from the skull cap and the base. When, however, our intention is to estimate the difference in any single case between cranial capacity and brain-volume, rather than for more general comparative purposes, it will be necessary to open the skull prior to opening the thorax so as to avoid emptying the venous sinuses, whilst at the same time the dura mater must not be stripped away from the skull cap nor the base be uncovered. With the object of estimating the amount of cerebral atrophy, a valuable series of observations were made by Dr. Hack Tuke, and the process he adopted is described as follows:— "The foramina at the base of the brain are carefully plugged with tenacious clay—that used by statuaries for modelling answers best; a small triangular piece of the frontal bone is removed with the saw; the calvarium is re-adjusted to the base, the dura mater being left attached. The space left by the attrition of the saw in removing the calvarium is filled with clay; and a narrow bandage with clay spread upon it, is made to surround the cranium three or four times, covering this space. If this manipulation has been carefully done, the cavity of the cranium will now be found as tight as a bottle. Sixty fluid ounces of water having been measured, a sufficient quantity to fill the cranial cavity is now poured into it by means of a funnel, through the orifice in the frontal bone, taking care that the stream does not wash away the luting of the foramina. The fluid which remains, after having filled

[1] On the weight of the brain in the different races of Man. Philos. Trans., 1868, pp. 506 and 526.

the cranial cavity is measured, and being deducted from the sixty ounces gives the amount employed. To this must be added half an ounce for the space occupied by the luting." Having thus obtained the cranial capacity, he deducts from it the brain volume, and obtains thus the exact measurement of shrinking or atrophy. Millet seed and sand have been used for measuring the capacity of the cranial cavity, and the latter in the dried skull has answered admirably. It must, however, be borne in mind that a fallacy may be introduced by the employment for this purpose of materials liable to be influenced by temperature. Fluids are, of course, more open to this objection, their expansion by heat being proportionately more rapid than solids, and hence it would appear that sand is preferable to water in these investigations. Mustard seed was employed by Professor Flower in his extensive series of researches.[1] The method adopted by myself arose from my employment of solid paraffin in obtaining casts of the brain surface, and the interior of the skull. It was soon apparent that the finest and most delicate impressions and most perfect casts could be obtained by the use of this substance, and that it did not share in the great disadvantage which accrues from the use of plaster of Paris, viz. that of great contraction during solidification. It is in this respect also far superior to white wax, which, as is well known, contracts much whilst solidifying. I proceed as follows:—Fill up the foramina at the base as previously described. A triangular or wedge-shaped piece is now sawn out of the occipital bone after removal of the calvarium, but retained in situ. The base is now filled up by melted paraffin, the skull cap replaced and fastened by luting, just as in Dr. Tuke's process, having previously trephined a piece out of the frontal bone. Through the latter orifice more of the paraffin is poured in until the cranial cavity is filled. When cool and solid remove the calvarium, as well as the wedge-shaped piece of bone from the occiput, and then gentle pressure from behind tilts the solid mass out of the cranium, when it will be found to form an exquisite mould of the interior. The mould thus obtained is

[1] The capacity of the crania contained in the Hunterian Museum of the Royal College of Surgeons was obtained by Professor Flower in this manner.

now to be measured by displacement, whence we obtain the cranial capacity. This method has afforded me great satisfaction, as it gives data of immense value at the expense of very little trouble: it supplies us with a method of—

1st. Estimating cranial capacity.

2nd. Gives us an exact mould of the cranial cavity.

3rd. As a permanent record—numerous linear angular measurement, and volume measurements may be obtained.

CRANIAL CAPACITY IN DIFFERENT HUMAN RACES.

CRANIAL CAPACITY.	Modern Parisians.	Parisians of 12th Cent.	Ancient Egyptians.	Negroes.	Australians.
Cubic Centimeters.					
1200 to 1300 . .	0·0	0·0	0·0	7·4	45·0
1300 to 1400 . .	10·4	7·5	12·1	35·2	25·0
1400 to 1500 . .	14·3	37·3	42·5	33·4	20·0
1500 to 1600 . .	46·7	29·8	36·4	14·7	10·0
1600 to 1700 . .	16·9	20·9	9·0	9·3	0·0
1700 to 1800 . .	6·5	4·5	0·0	0·0	0·0
1800 to 1900 . .	5·2	0·0	0·0	0·0	0·0

From some invaluable measurements by the late lamented Broca,[1] it was found that of 115 skulls of individuals living in the twelfth century, the *average* capacity was 1425·98 cubic centimeters; whilst of 125 skulls of the nineteenth century the average capacity was 1461·53 cubic centimeters. The accompanying table of percentage will be found of use by those interested in the subject of cranial capacity. It is given by Le Bon as illustrative of the relationship of race to cranial capacity.

[1] Quoted by Nogt, "Lectures on Man." (Anthrop. Soc.)

(*To be continued.*)

Critical Digests and Notices of Books.

On the Circulation of the Blood in the Human Brain. By PROFESSOR MOSSO. Turin.

(*Sulla Circolazione del Sangue nel Cervello dell' Uomo.* Roma, 1880. 4to, pp. 127.)

THE observations on which this important memoir is founded were made on three different individuals, two of them still surviving, whose histories are shortly the following:—

1. Catherine X——, a countrywoman, æt. 37, suffered in 1866 from syphilitic sores, which were followed by four successive attacks of secondary and tertiary syphilis, ending in 1875 in the destruction of nearly all the frontal bone, and a great portion of the two parietals. The removal of this large amount of bone left a large opening in the head, through which the pulsations of the vessels of the brain could be observed.

2. Giovanni Thron, an epileptic boy of 11 years of age, met with an accident at the age of 18 months, which destroyed a large part of the cranial substance in the right temporo-parietal region. So far as could be made out from the scanty history, it was not till eighteen months had passed that any important brain symptoms appeared; but at 3 years of age he began to suffer from epileptiform and epileptic seizures. When this boy came under Dr. Mosso's care in 1877, the elliptical opening in the skull measured 70 mm. by 35. On pressing firmly with the finger no pulsations could be felt through the hard membrane which covered the opening. The boy died, in Dr. Mosso's absence, of an acute gastro-intestinal catarrh, and at the post-mortem examination it was found that the cerebral lesion involved the posterior extremity

of the horizontal ramus of the Silvian fissure, the anterior part of the inferior parietal convolution (*lobulus supra-marginalis*), and ended in the middle part of the ascending parietal convolution. Inferiorly it extended as far as the three temporo-sphenoidal convolutions to a part of the angular gyrus. No motor paralysis was discovered during life.

3. Michele Bertino, æt. 37, in July 1877 was struck on the head by a brick weighing 3 kilogrammes, which fell from the hand of a workman who, at the height of 14 metres, was employed on the roof of the campanile of his native village. The lacerated contusion caused by this accident was irregularly triangular in shape, 7 centimetres by 4, and corresponded with the frontal region, of which the bone sustained a compound comminuted fracture. He was treated by a surgeon, who removed portions of brick and of bone from the wound, and who says that Bertino was quite conscious, and could speak when he saw him laid on a bed immediately after the accident; but Bertino himself, in his account of the occurrence, told Dr. Mosso that he remembered nothing, not even having received the blow, and that he believes he recovered consciousness about an hour after. After remaining twenty-four days in bed Bertino went to a neighbouring hospital to see another doctor, and in eight weeks from the occurrence of the accident he went to Turin. There was then a circular opening in the skull, of a diameter of 25 mm., in close proximity to the fronto-parietal suture. The general appearance of the opening, looked at from above downwards, was funnel-shaped, and the floor was formed by a red membrane with some granulations. When the patient stood erect, or sat, pulsations could be observed in the cavity synchronous with the radial pulse. There was an abundant secretion of pus from the cavity, whose capacity, when the patient was at rest, was about 5 cubic centimetres of water. In Bertino's account to Dr. Mosso he said that he found himself able to follow his occupation as usual about a month after he had left the hospital. The only difference he observed was, that when he put forth a special effort or stooped, he had a sensation as if his brain went upwards. In 1878 he had a fainting-fit; but in other respects he has remained quite well and like his former self, except

that he has become more timid and less courageous than before. The apparatus employed to measure the cerebral and brachial pulse consisted of various ingenious modifications of Marey's conjugate tympana, with special adaptations of gutta-percha plates, &c., as also of the cardiograph, sphygmograph, hydrosphygmograph, and plethosphygmograph—the last instrument, as the name implies, being used to measure the volume of the pulse.

As to the form of the pulse-curve, Professor Mosso says that the *tricuspid* pulse-tracing characteristic of the circulation in the brain can be produced in the radial and carotid arteries, and in the nasal cavity at will. This tricuspid tracing is so called because it shows three elevations, of which the central one is the highest, and the other two arise directly from the sides. After Landois, he distinguishes three parts in the tracing, the *ascending*, the *vertex*, and the *descending* part, and, after him also, elevations observed in the ascending part are called *anacrotic*, while elevations observed in the descending part are called *catacrotic*.

Dr. Mosso goes on to show that the tricuspid tracing depends on the state of the vessels. In fact, when a contraction of their walls is produced, an anacrotic pulse becomes a catacrotic. Figs. 6 and 8 show a tricuspid pulse in the forearm becoming catacrotic by cerebral activity. In Fig. 9 it is shown that when the "secondary elevation" (S) is non-existent in the radial pulse-tracing, it can be made to appear by such a contraction of the vessels as is induced by cerebral activity.

Fig. 7 shows how a tricuspid radial pulse can be converted into a catacrotic by inhalation of ammonia, at the same time that, as shown by the plethosphygmograph, the volume of the pulse was diminished by 16 cubic centimetres, and even by as much as 20 cubic centimetres. Figs. 10, 11, 12 deal with the changes in the pulse-tracing observed during fasting, and after eating. The general result is, that the absorption of food induces a catacrotic pulse-tracing, with a secondary elevation, as well in the radial vessel as in the brain itself.

Dr. Mosso brings forward the following considerations to prove that the tricuspid pulse is a phenomenon which depends on the state of the vessels. 1. The cerebral pulse-

tracing is often tricuspid at the same time that the carotid is catacrotic. 2. One radial tracing may be catacrotic, while the other is tricuspid. 3. A catacrotic radial pulse can be rendered tricuspid by heat, by cold, by muscular contraction stimulated by an induced current, and by voluntary muscular contraction, &c. Experiments are described to prove in detail these special points.

In Chapter IV. Dr. Mosso deals with the interesting question, "What are the effects on the pulse-tracing, of Thought and Emotion?" Fig. 21 shows that there was a large augmentation in the size of the cerebral tracings. 1. When mental activity was excited in Bertino by the question, how many eggs make twelve dozen? 2. By a reproof from Dr. Mosso to Bertino for having involuntarily moved his finger, so spoiling the radial tracing which was in the act of being compared with the cerebral. 3. By the clock striking when Bertino did not expect it; and 4. When Dr. Mosso asked him whether he was accustomed to say the *Ave Maria* at mid-day.

Dr. Mosso next proceeds to meet the objection raised by Professor Franck, that changes in the circulation might depend on changes in the respiratory movements. The author denies this for the following reasons:—

1*st.* Because often the forearm tracing shows diminution of volume during cerebral activity, while the respiratory movements remain unaltered in rhythm and depth.

2*nd.* Because we often find that the volume of the cerebral circulation increases during cerebral activity, while the respiration remains unaffected.

3*rd.* Because the characters of the sphygmic curve of the forearm show that a real contraction of the vessels takes place, which does not correspond with what is observed when a simple modification of respiration occurs; and

4*th.* Because we do not observe similar changes in the pulse of the brain and the forearm, which we should do if they depended on changes of the respiration, since respiration bears the same relation to the cerebral- that it does to the forearm-circulation.

Section 4 of this interesting chapter deals with examples of the law which Dr. Mosso believes he has established, that "the

emotion which is produced when passing from profound rest to a state of cerebral activity is always accompanied by a modification of pulse."

In Chapter V. the author deals with "Sleep in its Relations to the Circulation in the Brain." The author had formerly shown that in passing from the waking state into sleep there occurs a dilatation and relaxation of the vessels of the forearm, and that the act of waking is always accompanied by a contraction of the vessels. He has made experiments on natural sleep and artificial (induced by chloral hydrate) in the cases of Catherine X—— of Thron, of Signor Caudana, and of Bertino. The general result (to which there are, however, a few exceptions) is, that in the act of waking there occurs an augmentation in the size of the pulse-tracings. So much is this the case that frequently there is observed a similar augmentation, even when an unsuccessful attempt is made to wake the subject of experiment, such, for example, as calling him by name, but not so loud as to wake him. Dr. Mosso draws Fig. 28 to show that even the slight noise caused by his rising on his feet to call Bertino quietly (though the calling was not loud enough to wake him) sufficed to elevate the pulse-tracings a little. The tracing, besides, which was catacrotic before Bertino was called, became anacrotic afterwards, and remained so for some time. There were occasional augmentations in the size of the pulse-tracings which could not be explained (Table VI., line 26).

From Fig. 29, and also from several of the lines of Table VII., it appears that sometimes in the act of waking, the brain may undergo a diminution of volume, and may contain, in fact, less blood than during sleep, which, says Dr. Mosso, "suffices to render untenable the hypothesis which attributes sleep to a state of anæmia of the brain."

Chapter VI. deals with considerations regarding the nature of sleep, and of the phenomena which accompany it.

A modification of the circulation, it has been shown, takes place in passing from waking to profound rest, and from the latter to sleep. In sleep there occurs a dilatation of the vessels of the extremities which can be measured in the forearm by the plethosphygmograph. It corresponds with a relaxation

of the vascular walls. Every excitation from without causes a contraction of the vessels of the forearm and a subsequent increase of the blood-pressure, causing a larger flow of blood to the brain. On contraction of the vessels of the brain, which takes place on sudden waking, the general increase of pressure produces similarly a greater velocity of blood in the cerebral hemispheres. In sleep the heart movements become somewhat slower, and the changes above-mentioned, produced in the organism during waking hours by cerebral activity, are reproduced in sleep by external actions insufficient to wake the sleeper. Thus a sound, a voice, a touch, may modify the rhythm of the respiration, contract the vessels of the forearm, increase the pressure and flow of blood to the brain, and accelerate the rhythm of the heart; and yet the sleeper, on being wakened, asserts that he has no consciousness of having been affected by these causes. Dr. Mosso has called such influences, not depending on exaggerated respiration, and not leaving any trace in consciousness, *spontaneous*, and quotations are made from Carpenter and Maudesley in support of his view of them.

Just as was the case with the vascular supply to the brain, a noise, insufficient to wake the sleeper, may modify both the thoracic and abdominal respiration. Thus, in such a case the expiratory curve, which is a little delayed in time, is considerably increased in size when it does appear. After this there occurs a deeper and quicker inspiration; after which there takes place a slight commencement of expiration, and then a more marked and stronger expiration. On comparing the abdominal with the thoracic respiration, a want of correspondence between them is observed. When the thorax is making the expiratory movement the abdomen remains fixed; and when the thoracic inspiration begins, then takes place on the contrary an expiratory movement in the abdomen. But, during the continuance of the thoracic inspiration, elevation of the abdomen also occurs corresponding with inspiration. The cause of this is that, whereas during waking hours the diaphragm is one of the most active of the muscles, in sleep it becomes inactive and almost paralysed. During waking the abdomen rises with the elevation of the thorax, because the diaphragm falls synchronously with the movements of the

respiratory muscles. But when in sleep the diaphragm ceases to contract, dilatation of the thorax is accompanied by sinking of the abdomen, because the diaphragm is drawn up in inspiration like an inert membrane. When the sleeper wakes, the normal relations are speedily re-established.

The unconscious changes which occur during sleep are very remarkable, and, in the opinion of Dr. Mosso, with which Herbert Spencer's view mainly coincides, serve the purpose of increasing the blood-supply to the brain, and so of waking the animal when in danger. The animal which can bring most energy to bear in a short time has the best chance in the struggle for existence, since it may overcome an animal stronger than itself on the average, but which cannot put forth so much energy at once. In order to repair waste, and induce a high state of activity, animals must sleep profoundly; but during deep sleep a voice, a distant noise, a ray of light falling on the eyelids, or a slight touch, excites the respiration, contracts the vessels of the extremities, increases the energy of the heart's action, and the frequency of its beats, increases the blood pressure, and drives more blood to the brain; and thus the animal can pass from profound repose into complete activity in a time so short as to enable it to escape danger, which would overwhelm one whose processes were slower or less well adapted for their purpose.

In Chapter VII. is considered the occurrence of certain undulations in the pulse-tracings not dependent either on the respiratory movements (those so produced being termed *oscillations*), nor on the cardiac systole (called *pulsations*). These undulations are termed *spontaneous*, not in the strict sense of the word, of course, but in order to indicate that the cause of the phenomenon is not quite known. Dr. Mosso says they are due to changes in the elasticity of the vessels, and they are not the same as those which may be called *passive*, and which depend on an increase or diminution of the blood pressure without the vessels of the organ itself taking any active part in the formation. He concludes that the spontaneous undulations of the vessels constitute a phenomenon which has no relation, either with the innervation, or with the movements of the respiratory apparatus (Traube and Hering); that it is

produced without its being possible for us to believe that it depends on a certain venosity of the arterial blood (Cyon, Hering); on the accumulation of CO_2, or on the deficiency of O. The spontaneous undulations of the cerebral vessels have no constant rhythm, and the cerebral tracing may often remain for a long time horizontal with no other elevations than those of the pulse; and this even when, in the arm, respiratory oscillations are perceived.[1] The remaining sections of this chapter are occupied with delineations of the undulations in the tracings caused by electric irritation of the vagus in dogs, of those caused by slowing of the heart's action, and of the periodic variations occurring naturally in the carotid pulse, and of those induced by the action of chloroform.

In Chapter VIII. Dr. Mosso details some observations on vascular movements made on the ears of rabbits. He thought that previous observations might be vitiated by the emotion of the animals, and therefore constructed a cage with holes so placed as to enable him to watch the animals, without being himself observed. The results were quite in accordance with his expectation. He found that the contractions and dilatations which were described as taking place in the rabbit's ear, and which were ascribed to what Dr. Schiff called *the accessory heart*, ceased almost entirely when the animal was in complete repose. In profound tranquillity the artery of the ear would remain dilated sometimes for hours together. On the other hand, a whistle, a cry, the sound of the bells of the neighbouring church, the bark of a dog, and even a ray of light falling on the cage, all induced contraction of the vessels. His conclusion is that "the movements of the blood-vessels in the ear of the rabbit are related to the emotions and to the psychic state of the animal."

In Chapter IX. the influence of the respiratory movements upon the circulation of the brain and lungs is discussed. Speaking generally, it may be said that regular and easy

[1] May not these undulations depend, like so many other organic phenomena, on the fact that there is no stillness or sameness in life; so that life would be represented diagrammatically, not by a straight line, but by a wave? And may not the cause be that the cell, the ultimate element of organic structure, is itself in a continual swing of movement, now swelling and now shrinking, but never at absolute rest?—Tr.

respiration has so small an influence on the movements of the brain that it almost escapes observation. But careful observation discovers that in inspiration there is a rather lower pulse-tracing, and that in expiration the elevation of the pulse-tracings is increased. Not only is this so; but the elevation of the pulse-tracing is more or less proportionate to the depth of the inspiration and expiration, being higher than natural in the full expiration which succeeds a deep inspiration. In Fig. 49 the author shows an elevation produced in the cerebral pulse-tracings, 1st by a mental act (propounding to the subject of experiment the question how many are 6×45); and 2nd, by an involuntary deep inspiration. There is a difference between the elevations in these two sets of circumstances, however, which consists in this—that in the former the brain is increased in volume, while the circulation of the forearm is diminished; while in expiration both the volume of the brain and that of the forearm are increased, though the increase is proportionately greater in the brain than in the forearm, due probably to its richer blood supply.

As to the mechanical causes affecting the circulation during inspiration and expiration, little remains to be added to Haller's statement that "in inspiration the brain diminishes in volume because the blood recedes from the great vessels of the brain, but that in expiration it swells and increases in size because the blood is retained." Dr. Mosso details experiments which show that the jugular pulse (in a previous work shown to be the *negative* of the arterial pulse) disappears when the breath is held; and that the respiratory oscillations visible in the tracing of the forearm disappear when a pressure of 20 centimetres is applied to the surface of the whole arm.

In the lower limbs, on the contrary, there is an increase in volume during inspiration, and a diminution in the beginning of expiration. This is the case both in normal inspiration and in slower and deeper breathing. It is noticeable that a rapid diminution in volume takes place in the beginning of expiration.

The brain of Bertino was found to diminish rapidly in volume during a profound rapid inspiration; the systoles of the heart became more frequent, and the cerebral pulse

nearly disappeared. In the next inspiration the brain rapidly swelled; the systoles became less frequent, and the pulse very strong. After the expiration was finished a second diminution occurred in the volume of the brain, and a very small pulse was observed.

In this chapter Dr. Mosso describes a set of ingenious experiments made to determine the quantities of blood going to and coming from the lungs respectively, and the quantity of residual blood remaining. From these experiments, which our limits do not permit of our detailing, it appears that in inspiration more blood enters the lungs than issues from them; and in expiration the efflux is greater than the influx. A residuum of 15 or 16 cubic centimetres of blood was left in the lungs, and it was found that the blood did not issue from the lungs so rapidly as it entered.

Chapter X. deals with criticisms of various researches on the circulation in the lungs.

In his attempts to determine in Chapter XI. the influence of the respiratory movements upon the general pressure of the blood, the author agrees with Einbrodt and Ludwig "that the pressure obtains an increase during inspiration, inasmuch as each cardiac systole attains a tension greater than the preceding pulsation." When the blood-pressure was diminished by hæmorrhage, it was found that in inspiration the pressure descended; in expiration it was first raised and then descended. This was the opposite condition to the former. Comparing the state of fasting with that existing after a meal, it was found that after eating, the oscillations of the forearm pulse were more marked than in the fasting state, although the respiratory movements were less deep and full.

After inhalation of nitrite of amyl the elevations of the pulse-tracings of the brain are much increased, and the dicrotism is much more marked. In Bertino the carotid tracing, which normally consists of three elevations, of which the two last occur almost at the same level, tended under the influence of nitrite of amyl to eliminate the first of the two, while the second descended and became more pronounced.

The chapter on cerebral hyperæmia and anæmia is very interesting. Dr. Mosso says: "Were I asked which of all the

functions of organs is most dependent on even the slightest change which takes place in the material interchange, I should without hesitation reply, that consciousness is that function." The following experiment shows the prodigious activity of material interchange in the brain. On the 29th of September, at 1 o'clock, Dr. Mosso and Dr. Paoli made some observations on cerebral anæmia in the case of Bertino, by means of the gutta-percha plate and the hydrosphygmograph. Comparative tracings of the brain and arm were taken. Dr. Paoli applied pressure with his fingers on both carotids. The first two elevations were higher than before, but the third was much smaller, and the brain rapidly diminished in volume. After the 8th cardiac systole the pulse slowed notably, and in the brain was so small as to be scarcely perceptible. At the 12th pulsation, that is after about eight seconds of cerebral anæmia, Bertino was seized with an accession of convulsions; the face pale and eyes turned upward. Dr. Paoli immediately set free the carotids. As soon as Dr. Mosso saw that Bertino opened his eyes as if in wonder at finding himself in that place in that position, he tried without delay to continue the registration of the pulse: but the brain had undergone such an increase in volume, and the arm had moved so much that it was twenty seconds after the first onset of the convulsions before he could take up again the registration of the cerebral pulse, and a minute more before the forearm pulse could be registered. Seeing that there was nothing noticeable beyond a strong increase of the cerebral pulse, he suspended the observation.

Bertino asked them to proceed with their observations; but they thought it better not to do so.

Dr. Mosso comments on the remarkable dependence of the psychical functions of the brain on the interchange of its component parts. No other parts of the body manifest this to the same extent, not even the sense organs. We can produce an ischæmia of the whole arm, and literally expel the blood by an elastic bandage for twenty or even twenty-five minutes, without depriving the sensory apparatus of the nervous system of its power to transmit tactile calorific or painful impressions; while in the brain a diminution, not to speak of an arrest, of

the flow of blood for a space of time 300 times less is sufficient to abolish consciousness.

Some observations on the circulation of the blood in the sound cranium terminate this valuable memoir.

A. Rabagliati.

Des Contre-Indications à l'Anesthésie Chirurgicale. Par le Dr. H. Duret. 8vo., pp. 278. Paris, 1880.

In this highly able and instructive memoir M. Duret discusses the physiological action of anæsthetics on man and the lower animals, the causes of death as determined by physiological experiment, and by a review of all the reported cases of death from chloroform in man; the general and special contra-indications of anæsthesia in surgical practice; and the relative advantages and disadvantages of different anæsthetics. The work is so full of detail as not to admit of analysis within reasonable limits, and we would refer all who are interested in the important questions discussed to the original memoir itself. We would only briefly indicate M. Duret's generalisations as to the mode of action of anæsthetics. Anæsthetics may be administered in the great majority of cases with perfect safety. They cause temporary annihilation of the functions of the cerebral hemispheres and spinal cord, characterised by sleep, anæsthesia and muscular relaxation. The medulla oblongata alone continues its activity, and presides over the vital functions of circulation and respiration. During the whole period of narcosis, the activity of the medulla oblongata must be specially guarded. As a rule there are premonitory indications of the failure of this centre which must not be disregarded. But the medulla is liable to successive shocks which menace its vitality, and fatal results may ensue, though comparatively rarely, at various stages of the narcosis.

1. At the commencement of the inhalation, by reflex shock due to irritation of the respiratory passages.

2. At a later stage, before narcosis is complete, by bulbar shock, induced by the first waves of the anæsthetic vapours reaching the medullary centres and causing syncope and asphyxia.

3. In the later stages death supervenes by paralysis of the respiratory and cardiac centres. Usually the respiratory centres fail first, and asphyxia precedes syncope. But in certain morbid conditions the heart is the first to cease.

4. Lastly, death may be caused during the narcosis by traumatic shock, acting on centres already depressed and enfeebled.

After the narcosis death may result from general exhaustion of the nerve centres.

D. FERRIER.

Westphal on the Knee-Phenomenon. Berl. Klin. Wochenschr. 1881, No. 1.

As a contribution in answer to the question whether an anatomical lesion of the posterior root-zones is already present at a time when abolition of the knee-phenomenon is the only sign of the presence of tabes, Prof. Westphal relates a case of which the following are the chief points—Neuropathic inheritance; complete amaurosis three years before death; mental aberration requiring restraint at about the same period; trifling difficulty of articulation; knee-phenomenon found normal at various times during the progress of the case; no tabetic signs; three months before death, abolition of the knee-phenomenon on both sides; no other tabetic signs; death by intestinal complication. P.M.—Lesion limited to posterior root-zones, and posterior part of lateral regions; posterior roots and columns of Goll normal.

The author links the knee-sign with the lesion of the post. root-zones, not with that in the lateral columns, because on the one hand he has found after tabes with absent knee-phenomenon, lesion of the posterior root-zones, without lesion in the lateral columns, on the other hand he has found the lateral lesion in cases where the knee-phenomenon had been normal or increased.

Referring to the supposed uncertainty of the well-known tabetic test, he states that the cases called tabes, where knee- and foot-phenomenon were present, have been miscalled tabes, though there may have been ataxic symptoms present; he

says that such cases are presumably cases of insular sclerosis, and emphasises the statement that in no case of well-authenticated tabes is the knee-phenomenon present. With regard to its absence in normal persons, he states that he has never failed to find it present, even in cases of its pretended absence; still he admits that it may be absent if from any cause the tendon is not susceptible of the sudden extension, as from its shortness in thick-legged persons, or the presence of much fat beneath it. Further, the test may fail from excess or deficiency of the muscular tonus, which he regards as its principal condition. And finally, if the reaction were absent in an apparently normal person, he should think that that person is not normal, but the subject of latent spinal mischief; if, the reaction having been absent, it should reappear, he would be inclined to diagnose retrogression of incipient mischief, and to consider the fact as a hopeful feature in the history of a condition usually regarded as fatally progressive.

In conclusion, Prof. Westphal signifies his maintenance of his original assertion that the knee-phenomenon is not a simple reflex, but a complex manifestation, intimately related to a muscular tonus, which is possibly reflex. He considers that lesion in the posterior root-zone abolishes the knee-phenomenon by its enhancing or depressing influence on the tonus of the rectus muscle.

A. Waller.

Clinical Cases.

CASES OF ATHETOSIS.

BY FRANCIS WARNER, M.D. LOND., M.R.C.P.

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THESE cases came under notice at the East London Hospital for children.

Athetosis with Epilepsy.

CASE I.—Alice T., aged 13 years, was brought under my observation in January, 1880, complaining of a swelling in the axilla. This proved to be glandular, and soon subsided. The mother also complained that the left arm had been useless since she was six months old, that she was liable to fits, and rather stupid.

The girl was of fair stature, rather pale and thin, and of weak intellectual power. She could walk, assist her mother in the house, and answer questions, but was very dull and apathetic.

When seen, the following description was taken: *The left upper extremity* is almost useless for most voluntary purposes on account of the condition of the hand. If told to put her hand to her mouth, or to the back of her head, the hand gets there, being moved slowly and awkwardly by the action of the muscles moving the shoulder and elbow, but though flexion and extension of the elbow be sufficiently voluntary to perform such acts, involuntary movements of pronation occur, while the hand is being moved up to the mouth. All through the time of the general movement of this limb, it is obvious that there is much perfectly involuntary movement of the wrist, often causing marked flexion, or affecting independent digits.

As to the condition of the hand, the description was taken while the forearm was gently supported by the mother, the wrist being left perfectly free. It was then noted: The wrist droops, the metacarpus hanging straight down, but frequently supinated rather quickly, but not in a jerky manner, the hand then passes slowly back to its former position, the

series of movements being repeated. Flexion and extension of the wrist through a few degrees are nearly constant movements.

The movements of the fingers are almost incessant, the primary and quickest movements being flexor, followed by slower extension. The movements are slower than the jerks of chorea, and more deliberate, and looking like gesticulations; they are purposeless and gliding in kind.

The fingers are constantly going through grotesque movements, sometimes crossing one another, sometimes one is extended, while the others are flexed; there are no adductor and abductor twitches, such as are usually seen in chorea; the thumb is mostly turned in on the palm, but is often also extended. These finger movements appear utterly purposeless, looking as if the tendons were merely pulling upon dead fingers, as an automaton pulled by strings, or like the movements of a boneless limb, such as an elephant's trunk or an anemone's tentacle. The metacarpal bones are rather contracted together by the arching of the palm of the hand, giving it a narrow appearance.

The muscles of the forearm are neither hypertrophied nor atrophied. The heart and lungs were normal.

The following was the history of the patient.

She appeared healthy at birth: was suckled thirteen months.

When six months old, and while in apparently good health, she was taken suddenly in a fit, "as if choking, and wanting to be sick," there was some struggling, and the head was hot; the mother says that only the left arm and leg worked, and that the mouth was drawn to the left. After this fit she lay for three or four days quite helpless. There was no recurrence of fits for some months, then a second similar attack occurred, which was followed, after an interval, by a third paroxysm, each lasting about eight hours. There were in all three attacks during the first two years. There have been no fits from infancy till lately. The left arm has never been healthy since the first fit. The mother cannot say when the movements of the arm were first noticed, but they have certainly lasted many years, probably from infancy. It seems that she had some little use of the hand until the occurrence of the recent slight fits.

During the previous six or eight months epilepsy had returned, two or three slight fits having occurred each month, they set in suddenly, with loss of consciousness.

In the attack she would bite, look strange, and be powerless and stupid after them. Since the onset of the fits she has had less muscular power and more involuntary movements of the left hand as described.

The *patient* is the eldest of a family of six children, four of

whom are living, delicate children, but with no nervous symptoms. One child died in infancy of bronchitis.

The mother has had one miscarriage.

The *father*, aged 40, never had fits. He is now a teetotaller, because he finds that if he takes liquor it makes his head bad. Before the birth of this child he used to drink a good deal, and suffered from it. His mother was liable to fits for many years, falling, struggling, and biting her tongue. Her sister is insane.

The *mother*, aged 34, is liable to hysterical fits, not biting her tongue; she has two sisters, one aged 30, liable to epileptic fits, biting her tongue; the other 22, strong and well.

The patient was under treatment for one month. She took bromide of potassium, and the recurrent fits to a great extent subsided; the girl looked brighter, and the athetosis movements became lessened in degree.

As to causation, the early occurrence of fits in the child, and the inheritance of a tendency to brain disease on both sides of the family, point to heredity as the principal factor in producing disease. The purposeless, involuntary, and gliding character of the movements of the upper extremity were characteristic of athetosis.

Athetosis associated with Chronic Hydrocephalus.

Case II.—Thomas P., first seen, May 7, 1879. The mother complained of the large size of his head, that he could not stand, or sit up, or hold up his head; that he rolled his eyes about very much, and could not use his right hand.

He was then 1 year and 10 months old, obviously hydrocephalic, with a head measuring in circumference 23 inches, and from ear to ear over vertex 12½ inches; eyes much depressed, so that usually a portion of the pupil was hidden. The bones of the calvarium were thin and spread out or expanded, the anterior fontanelle was nearly closed. There were no signs of rickets; he had sixteen teeth. There was no otorrhœa. His general health was good, and no disease was detected in any organ but the brain. The condition of the right upper extremity especially attracted attention; the following description is compiled from notes taken on various occasions.

The limb was useless for any purposive movements; there was no voluntary power over the hand, and but little over the shoulder and elbow. When a flower was held up to the child, he looked at it and made a noise, evidently indicating his pleasure, then moved his left hand to the flower, took hold of it and tried to put it to his mouth. He could not take the flower with his right hand.

The wrist was frequently kept half flexed, while the fingers

were extended and flexed generally all together, in a slow and rhythmical manner. Pronation and supination were not common; the movements of the wrists were usually confined to flexion and extension of the fingers as described.

The hand was moved when pricked. There was no difference between the similar measurements in the two arms, but there was some rigidity of the right arm. The right leg was weak, but presented no athetosis. The child's birth was effected by instruments, and then it was noticed that the head was long and soft at the back. Convulsions occurred the day after birth, then no more till he was four months old, when he was strongly convulsed for four hours and greatly exhausted, but after a day or two he seemed as well as before. At eight or nine months old "he began to feel his feet," then his head was noted as enlarging, and he was frequently slightly convulsed. The mother is sure that before nine months old he could use his right hand, holding a crust in it, and that this power was gradually lost.

The child is still under observation (July 1880); no marked change has occurred except a few fits in June.

Here the condition of the limb was associated with brain disease and convulsions. The movements occurred in a paralysed arm, and were more limited in extent than in Case I.

Double Athetosis not associated with Epilepsy.

Case III.—Eliza R., aged 4½ years, came under observation May 13, 1880. She was a playful, intelligent, pleasing little girl, good and well-behaved, and soon became a general favourite. Her general health was good, and development appeared good in all particulars, but for the athetosis she was well nourished. Her habits were clean, there was no slobbering, and no paralysis, and she never had fits.

The child was unable to stand or do anything for herself on account of the movements of the hands and feet, associated with which there was no doubt some want of muscular power. The muscular condition of the head and face appeared natural. There appeared to be a fair amount of voluntary power over the shoulders and elbows, so that she could hold a doll to her. When told to take hold of a toy she brings her hand to it, but is unable to open the fingers so as to clasp it; all through the time of this effort the fingers are in constant movement. When the object is placed in her hand, she clutches it and is unable to drop it.

Supporting the left upper extremity free from the trunk by holding the humerus, it was possible to observe the following movements succeeding in a rhythmical manner.

The wrist was frequently bent backwards, and sometimes abducted. The thumb was mostly bent in on the palm, turned in under the index finger. The fingers were frequently extended at the metacarpo-phalangeal joints, remaining flexed at the inter-phalangeal joints. The greatest power of extension seemed to be in the index finger. In getting the hand near an object it moved about much before it came in contact with the object, and then could not grasp it on account of the condition of the fingers. The fingers were usually kept flexed; she could not spread them voluntarily. When the child is played with, her fingers spread open spontaneously, as also when attention is specially drawn to the other hand. When one's finger is slipped into her hand, her fingers grasp it, and cannot let it go unless the fingers happen to open of themselves. When she makes an effort and puts her legs out of bed to show her feet, the involuntary movements of the hands and fingers are increased, and movements of pronation and supination are noticeable.

The toes of both feet are continually being flexed and extended, but these movements are less in degree, and less characteristic in kind than those of the hands. She can kick her legs about in bed, but cannot walk.

The eyes and special senses are normal, and general sensation is good. Heart and lungs healthy.

The movements are very strange and purposeless, in kind they have more resemblance to voluntary movements than is usual in athetosis, and less of the gliding, successive-rhythmical character.

During sleep the hands are usually quiet.

This condition of the child appeared to have existed from birth. There were no signs of progressive disease.

The family were quite healthy.

Case IV.—The following case should probably be classed as one of Athetosis of the face associated with *petit mal.*

John Clark came under my observation, May 1878; he was then 12 years of age. The complaint made was that his hands twitched, his face worked much, and that at times he was quite silly.

He was a well-grown lad, of good complexion; his features presented a constrained appearance, and there was much movement of the face. The right hand twitched, but not violently. As he stood up there was some irregular muscular twitching all over the body. He spoke well, and was not himself conscious of the twitching of the face.

The movements of the face were very striking, and were carefully observed. They were principally confined to the parts about the eyes, nostrils and mouth, i.e. not affecting the forehead.

In July, when he was an in-patient at the East London Children's Hospital, I took the following description: Face: complexion somewhat anæmic, mucous membranes a little pale, not markedly so. There is considerable movement of the muscles of the face; he frequently closes his eye, next draws up the angle of the mouth on the same side, then contracts his eyebrows (corrugators), next he elevates them. He appears quite unaware that he is making grimaces. The inner portion of the occipito-fontales (grief muscles) frequently contracts in excess of other portions of these muscles; no movements of the hair or scalp seen.

The orbicularis oculi is frequently contracted, as indicated by the furrows around the eye, and the movements of the lower lid, while at the same time the eye is sufficiently open to allow of the pupil being distinctly seen (coincident of the orbicularis and levator palpebræ ?)

The eyes are much moved in a restless manner, and are frequently turned upwards when the eyelids are being closed. There is never any strabismus. Fundus oculi normal. The tongue is protruded at request, and kept out, and there is much irregular movement of its muscular structure.

As the patient stands upright with his heels together, there is scarcely any movement of the body. When he holds out his hands, and spreads his fingers, they are kept steady, but very slight adductor-abductor movements and almost inappreciable flexor-extensor movements of the fingers are seen; there are also slight movements of the toes. He walks well, with a steady gait, but slight irregular swaying movement of the head.

His heart and lungs appeared normal.

The following was the history of the case. He had always been strong and well till a year and a half or two years previous to my seeing him. He never had convulsious; he went to school when five years old, was bright and intelligent, and did as well as other boys till the autumn of 1876; never any complaint of pain till this time. About that date it was noticed that he made contortions of his face, and frequently nodded his head; then later on the fingers of the right hand began to work. After some months it was noticed that his manner was changed, he became forgetful and vacant—at times as if silly.

When sent on errands he went to wrong shops. He appeared at times to lose himself. One day he was found in the middle of the road at a distance from home, and could not say how he came there or where he lived. He became a tiresome boy, and at times passionate. If beaten he became worse, as if he had lost his reason entirely. In hospital he conversed well, and no mental failure was noticed.

Sleep was sound and refreshing.

As to the inheritance, the father and mother, and the families of which they were members, appeared healthy. The following is the account of the collateral members:—

(1) Male 25 years, healthy.	(4) Patient, age 12 years.
(2) Male 22 „ „	(5) Male 9, very healthy.
(3) 5 children died in infancy.	(6) Female 7, not strong.

The possibility of some defective inheritance is the only probable cause of the boy's illness.

When seen January, 1879, he went to school, was much less absent and forgetful; movements of face were less, principally consisting in contractions of both occipito-frontales muscles. No fits or attacks.

Now (August, 1880) he goes to school; seldom or never falls into "the lost condition." His face is peculiar, and somewhat impassionless, but without pathological movement except in the forehead. As he talks the skin of the forehead is frequently thrown into fine long transverse wrinkles by a slow movement.

Remarks.—These cases appeared to be worth putting forward for the opportunity of contrasting them.

Case I. presented athetosis associated with epilepsy, as is commonly the case. It increased and subsided in severity concurrently with the fits, and there was some mental impairment.

Case II. shewed hydrocephalus and hemiplegia, associated with athetosis and some rigidity. The movements were less in degree than in Cases I. and III.

Case III. presented double athetosis, or double hemikinesis. The cause existed from birth, and the child was free from epilepsy.

Case IV. is classified as athetosis with some reserve; but the persistent rhythmic contractions of the facial muscles, the long continuance of the movements, and general steadiness of gait, suggest a greater alliance to this condition than to chorea. The cases agree in the purposeless, gliding character of the movements which affected principally the upper extremity, and were most marked in the digits, and in the flexor-extensor movements of the wrist, less marked in pronation-supination, and least in the elbow and shoulder.

CASE OF PARETIC DEMENTIA: INTERCURRENT ATTACK OF LEFT-SIDED CONVULSIONS, BEGINNING IN, AND CHIEFLY CONFINED TO, ARM AND FACE; LESION OF POSTERIOR EXTREMITY OF RIGHT SUPERIOR FRONTAL CONVOLUTION.

BY RINGROSE ATKINS, M.A., M.D.

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THE influence of localised destructive lesions of the motor area of the cortex, and of the subjacent medullary fasciculi, in the production of limited paralyses (crural, brachial, facial, and their associations), is now fairly established by the results of combined clinical and pathological observation; the corresponding effects of irritative lesions of the same area, though recognised and placed on an almost equally firm basis, by the results of experimental physiology, have not, however, as yet received the same amount of extended and definite support, from the experiments of disease, partly from the spreading character of such lesions generally, and partly from the complex train of symptoms to which they give rise; and therefore the particulars of the following case, though not as satisfactory as regards the absolute limitation of the convulsive movements as could be desired, or as is necessary for the purposes of such an inquiry, may yet be of sufficient interest and importance to warrant my recording it, pointing as it does to the localisation of the gross movements of the arm in that region in which experimental investigation has indicated them to be.

John Reilly, formerly a member of the Royal Irish Constabulary, aged 44 years, admitted into the Waterford Asylum May 25th, 1878. The patient, who was unmarried, was accompanied to the Asylum by his brother, from whom it was ascertained that there was no hereditary history of mental or nervous disease, so far as he knew; there was no personal history of syphilis, sexual excesses, or intemperance, the man having been an efficient member of the Force, until struck down by disease. Eighteen months before admission he had had an attack of right hemiplegia and aphasia, from which he

quite recovered; this was, however, followed by a second similar seizure some time after, and again recovery ensued, the man regaining his full powers of locomotion, and, as is stated, of speech also. Five months previous to admission it was noticed that his memory began to fail and his mind to wander, while his utterance became thick and partially indistinct. Gradually he became irritable and developed delusions of persecution, fancying that those around him had conspired to murder him; as a consequence of these delusions he became at times violently excited and noisy, calling loudly at intervals for the police, and in the end becoming so unmanageable and dangerous to those around him, that he had to be brought to the Asylum. When examined immediately on his admission, he was, *mentally*, perfectly quiet and calm, his condition of mind being characterised by dulness, apathy, and apparent indifference to his position and surroundings: he showed no inclination to enter into conversation, and never spoke unless spoken to, and then invariably replied that he felt quite well, and that there was nothing amiss with him; he did not then give expression to any delusion. His facial expression was weak and languid-looking, and the angles of the mouth were slightly drooped. He stood firm and erect, and there was no *motor* disturbance; he walked without any dragging or incoordination of either limb, could stand well with his eyes shut, and turn round when desired without staggering much. The grasp of the right hand was not as powerful as that of the left, but the difference was not great. When replying to questions, there was a noticeable tremor and jerky action of the lips, and he evidently found difficulty in completing his words, as the last syllables and latter words of a sentence were drawled out and jumbled together: the tongue was affected with tremor and spasmodically protruded and retracted. The pupils were equal and normally motile. There was no apparent defect of vision or hearing. The condition of *tactile* sensibility was not investigated, as the degree of dementia was too deep to allow of reliable results being obtained; he had, however, two sores over the buttocks, which he did not appear to feel, or hardly to be conscious of, so that it is probable that there was analgesia to some extent at any rate. His general muscular development was good. During his residence in the Asylum the mental powers rapidly failed, the loss of memory increasing and the dementia deepening, while the articulatory defect steadily progressed. With this, however, there was no decided loss of motor power in any of the limbs, beyond a general muscular weakness and unsteadiness; and he was in this condition on the 23rd of September, 1878, when, at 11.15 A.M. on that day, he was suddenly seized with unilateral left-sided

convulsions, and became unconscious. The spasms—which were clonic—began in the left arm, as a whole, and in the left side of the face (angle of mouth and forehead), and travelled downwards to the leg, which was much more mildly convulsed. Soon after the spasmodic movements commenced in the same order at the right side, but were neither so powerful nor continuous, soon ceasing there and in the left leg. The left arm and left side of face continued, however, to work at very short intervals, the movements involving the entire arm from the shoulder down, the limb being jerked up and partially thrown across the chest, the angle of the mouth twitching at the same time. At 12 o'clock, the spasmodic movements continuing uninterruptedly, forty grains of bromide of potassium were administered, and at 3 o'clock, but little abatement being noticeable, another dose of forty grains was given (note made at 3.30), and at 4.20 P.M. the fit ceased, and he passed into a heavy sleep, in which he lay at 11.20 P.M. The next day it was with difficulty he could be roused, and for some days after he was drowsy, and the dementia had perceptibly deepened. From this time there ensued a more distinct loss of motor power, the left side being chiefly affected, and especially the left arm; the speech defects also rapidly increased, the lips trembling and jerking to a marked degree whenever he attempted to speak, which he rarely did: he used to keep the jaws tightly clenched, and was almost constantly grinding his teeth. He had no further convulsive attack, but becoming more and more weakened and demented, he sank and died on January 3rd, 1879.

Autopsy forty hours after death.—The body was fairly nourished, the calvarium dense and solid throughout, the diplöe being replaced by compact bony tissue. There were no morbid adhesions between the dura mater and the inner surface of the skull. The membrane was unaffected, its vessels posteriorly were filled to repletion with blood (hypostatic congestion), and it was attached by a few filamentous bands to the pia mater at several spots parallel to the great longitudinal fissure. The pia-arachnoid was somewhat opaque, especially where it bridged the sulci, being here of a milky colour. At a spot corresponding to the posterior extremity of the superior frontal, and the summit of the ascending frontal convolutions of the right hemisphere, the membrane was distended into a bulla the size of a walnut, by a collection of fluid beneath; and a similar bulla, but much smaller, occupied a more anterior position on the left superior frontal convolution. The membrane enclosing the fluid was thin and pellucid, and on being ruptured the contents were found to consist of a clear reddish-brown serum. On carefully removing the pia mater from the

spots thus affected, a deep atrophic depression, large enough to admit the tip of the thumb, was found involving the posterior extremity of the right superior, and portion of the summit of the right ascending frontal convolutions; and a slight depression existed beneath the bulla on the left superior frontal. There was no erosion of the surface, or softening of the substance of the affected convolutions, the cortex being apparently only depressed into a pit-like hollow. The pia mater, though adherent to the cortex in small patches scattered over the fronto-parietal convolutions, was not so at the atrophied spots, the membrane stripping readily. There was no gross lesion in any other part of the cortex, and no morbid conditions discoverable by the naked eye existed in the medullary substance or central ganglia. The vessels at the base were slightly atheromatous. Portions of the atrophied and neighbouring convolutions, together with the pons medullæ and a piece of the cerebellum, were placed aside; and having been hardened in iodised spirit and bichromate of potash, prepared sections of the atrophied gyri showed the following morbid conditions:—The cortex did not appear to the naked eye to be notably thinned. The walls of its *vessels* were greatly thickened by a closely-set nuclear overgrowth, so that their laminæ were contracted often to a very considerable extent. Viewed in transverse section, the fibrous coat of the arterioles, thickened and wavy, remained in some instances otherwise intact, the aggregation of nuclei surrounding it and distending the investing sheath; while in other instances the nuclear growth seemed to have taken possession of the entire thickness of the wall, either obscuring or altogether replacing the normal tissues. Viewed longitudinally, the surface of the arterioles appeared to be covered over closely by the nuclei. The perivascular spaces were comparatively clear and did not contain scattered nuclei, particles of hæmatoidin or granular débris. Sections through the adherent pia mater and cortex showed the membrane to be much thickened, its meshes infiltered with nuclei and the remains of minute blood extravasations, the walls of its vessels coarse and thickened, though not nucleated in the same way as those in the subjacent cortex, and their lamina filled with broken-down blood-corpuscles. Where firmly adherent to the cortex, the latter seemed to be continuous with the membrane by a fine granular material in which the entering vessels were embedded; and where removed purposely, or in the preparation of the sections, the cortical surface was irregular and overlaid by a refractive yellowish material like disintegrated blood. The *nerve-cells* in all the sections examined seemed to be similarly affected, any difference being one of degree; the different cortical

laminæ appeared to be compressed, and the first layer was coarse and molecular-looking, and studded with many free nuclei and ovoid refractive bodies (artificial products?). The larger corpuscles of the third layer were diminished in number in patches, a group appearing, and then a blank occurring, with but one or two; many of those remaining had lost both basal and apical processes or basal only, the angles being rounded and the entire corpuscle surrounded by a clear space. Few were deeply pigmented, or in an absolutely granular condition. The majority of the smaller cells seemed to be unaffected. The *neuroglia* throughout was coarse and molecular, containing lymphoid cells, granular bodies, and refractive spots.

The partially localised convulsions recorded above were clearly intercurrent in the course of a case of general paralytic dementia, of which this was, I believe, an example of that dubious form of the disease met with in this country; and it is a question, of course, how far the cortical lesion can be considered as playing a part in the causation of such seizure. Unilateral or partial convulsions, as is universally known, are of common occurrence in the course of general paralysis, though it was not until a comparatively recent period that their cortical origin began to be recognised and investigated; and even now, while it is conceded by the most recent authorities on the subject that the localised adhesions of the pia mater to the cortex in the motor area are the pathological factors in the production of the limited convulsions and spasms, there are not, I believe, as yet many cases on record where any single lesion of this kind, in any definite motor centre, has been directly associated with any limited convulsive seizure in the relation of cause and effect. In the present instance the most pronounced and definite cortical lesion, though not an adhesion, occupied a position closely corresponding to that which Ferrier has experimentally shown to be concerned in certain movements of the arm, and which is supported by the facts of human pathology. He quotes a case recorded by Lépine, of left hemiplegia followed by convulsions limited chiefly to the left arm, in which a small hæmorrhage was found at the posterior extremity of the first frontal convolution of the right hemisphere; and another, by the same observer, of left hemiplegia, in which convulsions occurred, either limited to the left arm or invariably commencing there; a yellow patch was found at the bases of the first and second frontal convolutions of the right hemisphere. Though not strictly analogous to these cases, either in the sequence of the symptoms or in the nature of the lesions found *post mortem*, the observation which I have recorded possesses, in common with them, the

essential features of limited convulsions and localised cortical disease, and hence it may be regarded as bearing on the question at issue. It may, perhaps, still be asserted by some, that such convulsions coming on in this way cannot always be traced to their origin, that they occur independently of gross or even of discoverable microscopic changes in the brain structures; but though this is doubtless true in certain cases where the seizures are of a more distinctly epileptic type, yet I think that their occurrence once in the course of such a case, the limited character of the convulsions at the beginning and termination of the attack, and the duration of the spasmodic movements, point strongly to their purely organic origin; and though the lesion itself may not, when discovered, have presented the characteristics of an "irritative" one, yet having regard to the time which elapsed since it had been active and the subsequent changes which may have occurred to alter its appearance, the position it occupied should, I think, be considered rather than its exact nature. So impressed was I, indeed, at the time with the character of the convulsive seizure, that I ventured the opinion that it might be possibly due to some lesion in the motor area of the cortex; and any other condition found failing to account for it, the morbid alteration affecting the posterior extremity of the first frontal and the summit of the ascending frontal gyri must, I believe, be credited with a causal influence in the production of the limited convulsions.

A CASE OF INJURY TO THE LEFT FRONTAL LOBE OF BRAIN, FOLLOWED BY AN ABSCESS.

BY HENRY ASHBY, M.D. LOND.

Physician to the General Hospital for Sick Children, Manchester.

J. H. W., aged 6 years, was admitted to the hospital Jan. 4, 1881, with the following history. He is the youngest of nine healthy children, and was well till July 26, 1880. On that date, while he was playing with his sister, he fell forward upon a sharp pitchfork she held in her hand, the prong striking him above the left eye. No external wound resulted, except an abrasion of the skin, the upper eyelid became much swollen, and he suffered much pain in the forehead and ears. During the next few weeks he was irritable, thirsty, feverish at night, and frequently vomited. Just before Christmas his sight failed him, in consequence of which he was brought from Huddersfield to the Manchester Eye Hospital for advice, and was transferred to the children's hospital.

On admission.—He is an irritable child, fairly well nourished, talks with a nasal twang, and is apparently somewhat deaf, or dull of comprehension. There is no mark of any wound about the left eye, but he screws it up. He is quite blind; the pupils are dilated, but act sluggishly. There is slight but well-marked right facial paralysis.

He lies in bed with knees drawn up: he can use both hands, but the right is the weakest; on being told to squeeze one's hand, he exerts some power, then says "I can't," and cries. He can walk fairly well, but is weak on his legs. All the superficial reflexes well-marked. Knee reflex, present not exaggerated; no rigidity of the limbs. Both optic discs swollen; edges indistinct; veins tortuous and full; arteries only seen on discs here and there as fine threads; several yellow spots on discs obscuring vessels, and others glistening in retina, near disc (hæmorrhages).

He remained much in the same condition, being irritable, fretful when touched, and sometimes crying out at night, and passing his urine and fæces into the bed.

January 28th.—Had a fit this morning, lasting twenty minutes, clenching his hands, and flexing limbs; he became comatose, and died in the evening.

Autopsy.—Forty-eight hours after death. Head only examined. On removing skull and dura mater, it is apparent that the convolutions of the left frontal lobe are dry on the surface and much flattened out, the whole width of the lobe being increased. The ascending frontal, superior, middle, and inferior frontal convolutions are involved in the flattening; the ascending parietal in lesser degree.

On gently raising the frontal lobe from its bed, it is evident from its elastic feel that it contains fluid, and from the yellow tinge of the inferior surface that this fluid is pus. It is adherent to the centre of the internal surface of the roof of the orbit, at a spot the size of a threepenny piece. On removing the brain and examining the bone, there is a hole through the roof of the orbit, large enough to admit the tip of the little finger, directed backwards and upwards. On section of the frontal lobe, it is found to contain four to five ounces of thick greenish pus; the inferior wall of the abscess is not much thicker than cardboard; the pus has evidently exerted considerable pressure on the inferior, middle, superior, and ascending frontal convolutions, flattening them against the roof of the skull, and involving the white substance immediately beneath them, but leaving the whole thickness of their grey matter. Examination of the optic discs shows that they are prominent, and the veins are full and tortuous. The optic sheaths behind the eyeballs are in a semi-distended state. A naked eye examination of the nerves, between the fundus and chiasma, after hardening in ammonia bichrom. and spirit, shows them to be swollen (in comparison with a healthy nerve). Longitudinal sections of the fundus, examined microscopically, show the disc much swollen, increase of nuclei, small collections of nuclei around arteries, and the lymph channels between the strands of the nerve, much distended. Transverse sections made at intervals to chiasma show these enlarged lymph-spaces in the nerve for $\frac{1}{3}$ to $\frac{1}{2}$ in. behind fundus; nearer the chiasma the nerve was apparently normal. Both nerves exhibit these changes.

Remarks.—In this case it appears that the sharp, narrow prong of the pitchfork entered the orbit, between the upper eyelid and eyeball, pierced the roof of the orbit, and entered the frontal lobe for a distance apparently of at least an inch, judging from the size of the opening through the orbital plate. This injury was not immediately fatal, but was followed by a local inflammation, which ended in an abscess that

proved fatal in six months. The abscess totally destroyed the convolutions on the under surface of the frontal lobe, pushing inwards and undermining those on the inner side, thrusting upwards and flattening out the ascending, superior, middle, and inferior transverse, against the roof of the skull, completely destroying the white matter underlying them, and leaving the whole thickness of their grey substance, but in a state of compression.

Notwithstanding this state of things, the boy could walk to hospital, his memory and general intelligence was good, and there was no loss of speech. But he was somewhat deaf; had slight facial paralysis on the opposite side, and some weakness of the right arm; troublesome pains along the facial divisions of the fifth, and loss of control over his sphincters.

PAIN IN THE OCCIPUT AND BACK OF NECK. STRONG RETRACTION OF THE HEAD. GLIOMA OF THE RIGHT TEMPORO-SPHENOIDAL LOBE. NO DISEASE OF CEREBELLUM.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

THE patient was a gentleman aged twenty-seven. In 1873, as I am informed by his medical attendant, he complained of what he called "giddiness" or "nervous attacks." He subsequently spent several months in North America, engaged in sporting. In 1877 his symptoms were nearly the same as in 1873, and a physician who saw him regarded these and the former attacks as those of *petit mal*. He never lost consciousness. During last autumn he spent the season in successful grouse shooting. A week before his death he hunted on one day from ten to six o'clock. During the last two months of his life he complained of severe pain over the occiput and back of the neck, extending upwards on the right side of the head to the forehead and sometimes down the neck. The pain generally came on during the night, and disappeared after breakfast. He was sometimes free from pain for several days together. There was occasionally a little sickness. In the paroxysms of pain the head was drawn back during the last few weeks of his life, and he complained of hemiopia. There was very slight facial paralysis of the left side.

Autopsy.—There was nothing abnormal in the appearance of the cerebral surface on opening the skull. When the brain was removed and turned upside down, it was noted that the sulci of the inferior surface of the right temporo-sphenoidal lobe were effaced. Palpation of this lobe was accompanied by a sense of fluctuation, in striking contrast with the corresponding lobe of the left side, which presented the ordinary resistance of healthy brain. A vertical section through the right temporo-sphenoidal lobe showed that the grey matter of the cortex had disappeared, the exposed walls of the section presenting a flesh colour. The structure was, in parts, of a consis-

tence somewhat more dense than that of healthy medullary matter which, however, it a good deal resembled in appearance. At the depth of rather less than an inch the knife passed into a cyst-like cavity containing about two drachms of straw-coloured fluid, and large enough to hold two or three walnuts. The walls of the cavity were very smooth, and consisted of the same flesh-coloured substance as had been previously cut through. Several small hæmorrhages were observed in the walls of the section. The abnormal structure or tumour merged quite insensibly into the surrounding brain substance, which was more or less softened throughout the whole of the right temporo-sphenoidal lobe, and some distance into the occipital lobe, as well as, though to a less extent, into the lower part of the convolutions bordering the fissure of Rolando.

The cerebellum presented no signs of disease in any portion of its structure, nor was there any appearance of flattening of its upper surface. Under the microscope the tumour presented the characters of soft glioma tending to myxoma, numerous protoplasmic cells, with a few much larger ones, containing many nuclei, and bearing a general resemblance to mucus globules.

The patient was only seen by me shortly before his death, when he was in a state of coma, which was sometimes profound, whilst at other times he could stand, talk, and swallow liquids, although with great difficulty. The coma was partly due to a dose of morphia, which had been given for the relief of severe pain. The head was retracted, so that the occiput was nearly touching the cervical portion of the spinal column, the eyes were upturned and divergent, there was slight facial paralysis of the left side. He would keep his right hand to his forehead, and when asked if he was suffering pain in the head, would signify that this was so.

I learned that for upwards of two months past he had been subject to daily attacks, lasting many hours, of severe pain about the back of the head and neck, accompanied occasionally, but not often, by vomiting. In these attacks the head was drawn back, I was informed, in the way which I have described. Impairment of sight had been complained of, and there had apparently been hemiopia. An ophthalmoscopic examination had been proposed, but not made.

Incomplete as is the case, it seems an important one to place on record, because of the very striking symptom, retraction of the head.

Dr. Hughlings-Jackson has recorded examples of what he calls "cerebellar rigidity (part of which is retraction of the head), in cases of cerebellar tumour. In some of these there were,

besides, paroxysms like those of surgical tetanus. Similar observations have been made by Dr. Stephen Mackenzie. In the 'Lancet,' January 24, 1880, Dr. Hughlings-Jackson writes: "I have only once seen seizures like tetanus in a case of local cerebral disease, and in that case they occurred during the last hours of life—during dying." In the paper referred to he records a case of retraction of the head with tumour of the cerebellum. Of course retraction of the head is known to occur from other pathological changes, as cerebro-spinal meningitis for example, but for purposes of localisation cases of tumour supply the best evidence, though, unfortunately, from their size this evidence is not often very definite.

It is remarkable that a person suffering from so extensive and grave an intra-cranial lesion should have been able to enjoy a long day's hunting within a week of his death.

Abstracts of British and Foreign Journals.

On Hydrops Articulorum intermittens.—Under the above heading Dr. Seeligmüller has published in the *Deutsche Medicinische Wochenschrift* (1880, Nos. 5 and 6), a description of a case of this obscure and interesting affection, followed by an analysis of 12 observations of the disease made by other authors, this being the complete literature of intermittent swellings of joints. Seeligmüller gives the following definition of the disease: "An otherwise healthy adult is affected in regular intervals, generally without any prodom and without discernible cause, by *an intense swelling of one or both knee-joints, there being no signs of inflammation or fever.* The swelling reaches, after a certain time, its maximum (stadium incrementi), remains there for a while on the climax (stadium acmes), and disappears likewise after some time, without leaving the slightest alteration in the affected joint. (In Seeligmüller's own observation, however, this was not the case; the left knee was always a little swelled and painful to the touch.) *In most cases the swelling returns with mathematical precision on a certain day*, sometimes even at a fixed hour, so that the patients can foretell the outset of the affection."

Since the publication of Seeligmüller's article, three other cases have been reported in the same journal by Pletzer (No. 3 f. 1880), Fiedler (No. 3, 1881), and Kolbe (No. 4, 1881).

Concerning the *etiology* of the affection, out of 13 cases collected by Seeligmüller (including his own observation) there were 7 females and 6 males. The three cases published since were all females, my own patient (of which I shall speak further on) also female. This gives a total of 11 females to 6 males.

The *age* of the patients was between 12 and 54 years; my patient was 9 years at the time when the disease was first noticed.

As to the *cause* of the affection, in two cases there is a history of intermittent fever. Kolbe's patient is evidently hysterical. In his, as in two other cases, an injury of the knee must be considered as the immediate cause of the disease.

The intervals between the fits are very variable; 8 days, 11 days, 13 days, 4 weeks. In my case they are absolutely irregular. The case reported by P. Bruns is most remarkable in this respect, as the patient had the swelling at regular intervals of 12 days during 8 years. In many cases there was an absolute intermission of months, and even years, after which the affection began again, sometimes adopting a new type. The duration of a fit is about from 3 to 8 days; in my case sometimes 2 to 3 weeks.

In 9 out of Seeligmüller's 13 observations, the knee-joint was the only articulation affected by the disease; for this reason it was always described as hydrops genu intermittens. Twice there is no particular mention as to the seat of the swelling. One patient (Löwenthal's case) began with swelling of the right elbow. In Seeligmüller's case, the left knee and one of the hip-joints showed the swelling. In Fiedler's case, as well as in mine, nearly every articulation of the body is occasionally the seat of the disease, but there is usually only a single joint affected in each attack, sometimes two at a time.

There is no notice of any *secondary alteration* of the joints, not even in Seeligmüller's case of 30 years' standing.

The history of my case of Hydrops articulorum intermittens is this:—

Marie M., æt. 11, has been suffering for the last two years from swelling of one or another joint, combined with extreme pain, hyperæsthesia of the skin in the neighbourhood of the swelled joint, consequently entire loss of function during the attack. The onset of the swelling is as sudden as a stroke of lightning; no symptom of any kind precedes the attack. For instance, the child is writing; suddenly she feels a violent pain in her wrist, and is unable to finish the word that she was just going to write down. The swelling increases more or less during the next 2 or 3 days; it remains in the stadium acmes for a variable time, and finally disappears gradually.

At first the attacks occurred with intervals of several weeks; but, by degrees, they came more frequently and lasted longer. Since February 1880 there were only intervals of a few hours, so that the little patient could hardly ever leave her room. The pain was so great that even when only a joint of an upper extremity was affected, the girl scarcely ventured to walk about in the room, because the commotion caused thereby increased her suffering. The disease has some seats of predilection, viz. the wrist, knees, and the ankle-joint; but all articulations of the extre-

mities, and sometimes the cervical vertebræ, suffer occasionally. Fever or any other disturbances of general health were never observed.

The *treatment* consisted at first in anti-rheumatic remedies: salicylic acid, quinine, iodide of potassium, colchicum; hot steam-baths were followed by an evident increase of the disease, while other remedies had no effect of any kind. Several other drugs were tried with the same negative result. The first thing which seemed to be of some use was arsenic. While this remedy was given, the child had an interval of 6 days; but it must be mentioned that she was then staying in the country for a few weeks and could be in the open air nearly all day, so that this may partly account for the improvement.

I saw the patient for the first time on the 18th of August, 1880, and began at once with applications of the galvanic current. I omitted to say that faradic electricity had been used in the beginning of the illness by the father of the child, who is a physician in Dresden. At first it seemed to alleviate the pain a little, but after awhile it failed to do so, whereas the application of an ice-bag had a better, though but a temporary effect. In examining the child I stated that the local use of a faradic current *increased* the pain, without in the least relieving the swelling. This is a remarkable fact with regard to the distinction of this disease from rheumatic inflammation of the joints. In these cases, as has been shown by Drosdoff, Abramowosky, and frequently confirmed by myself, there is a considerable diminution of the faradic sensibility in the skin of the affected parts, so that the strongest currents may be applied without pain; besides faradisation is always followed by more or less alleviation of the pain and swelling.

My treatment consisted in the application of a moderately strong galvanic current to the cervical region of the spine during five minutes daily. I intended to act on the vasomotor centres situated in those parts. The effect of this proceeding was decidedly good. Within a few weeks the free intervals became longer; the swelling, when it occurred, was considerably less intense, the pain tolerable. The child began to go to school, which she had not been able to do for several months. In January there was an interval of 22 days, since then till now (March 5th) the patient has had two very slight attacks, and lasting a few days. I must not forget to add that since the end of November the child recommenced to take arsenic, but in very small doses (3 drops of Fowler's solution in a day). I think it may be hoped that a per-

severing use of the galvanic current, occasionally combined with arsenic, will cure the little patient of her obstinate and troublesome complaint, and if a conclusion ex juvantibus be permitted, the undeniable action of the electric current cannot fail to support the idea expressed by Seeligmüller and other authors, that Hydrops articulorum intermittens must be considered as a chronic affection of the vaso-motor nerves.

R. H. Pierson, M.D.

Löwenfeld on Electro-Therapy of the Brain.—Löwenfeld (*Centralbl. f. d. med. Wissenschf.* No. 8, 1881), gives a preliminary account of the results of experiments he has made on rabbits and other animals as to the effect of passing electrical currents through the head longitudinally or transversely; and also of placing one pole on the exposed brain and the other on the neck, according to the method of Legros and Onimus. He finds—

1. A descending current (+ on forehead, – on neck) causes contraction of the vessels of the pia mater.

2. An ascending current (+ on neck, – on forehead) causes dilatation of the vessels.

3. In transversely directed currents, there is dilatation of the vessels in the side of the anode, and contraction at the cathode.

4. Induced currents, longitudinally or transversely directed, cause hyperæmia of the brain. This point requires further investigation, but it seems as if the induced current is not limited to action on the vessels.

Gombault on the Segmentary Periaxillary Neuritis.—In the *Archives de Neurologie*, No. 2, 1880, Gombault finishes his paper on segmentary neuritis, the first part of which was noticed in 'Brain,' Part XI. p. 423. Gombault is of opinion that the periaxillary neuritis may result in Wallerian degeneration when the inflammatory process is sufficiently intense to cause rupture of the axis cylinder. This occurs from a participation of the axis cylinder itself on the inflammatory action, not by mere pressure of inflammatory products. He draws an analogy between the periaxillary neuritis proper, and the Wallerian degeneration which may accompany it, and the changes which Ranvier has shown to occur in the proximal and distal ends of a divided nerve respectively. In the former there is a parenchymatous neuritis, in the latter there is degeneration in consequence of the separation of the nerve from its trophic centre. In traumatic neuritis in man the changes are such

as are seen in periaxillary neuritis. In chronic anterior polio-myelitis the question is whether the changes which occur in the nerves are merely passive, and merely the result of the disappearance of the multipolar cells, or whether there is active irritation or inflammatory action. The latter is the view advocated by Charcot, and Gombault is of opinion that the facts are in favour of it. Periaxillary neuritis exists, and the irregularity of the distribution of this process sufficiently accounts for the appearance of degeneration at different levels in different nerves.

As regards the general action of lead on the lower animals it appears to induce all the principal phenomena of anterior polio-myelitis. The sensory nerves do not seem to be affected. In the spinal cord the anterior horns alone are affected. The multipolar cells are in large measure vacuolated; the neuroglia and walls of the vessels indicate products of inflammatory irritation. In the muscles also of guinea-pigs poisoned with lead, very delicate fibres are visible, not met with in normal guinea-pigs.

Debove and Boudet on the Pathogeny of Tremors.— Debove and Boudet de Paris (*Archives de Neurologie*, October 1880) apply the results of an investigation of the characters of the muscle curves in healthy individuals under varying conditions to the explanation of tremors.

The sudden elongation of a muscle excites it to contraction (the tendon phenomenon), and this may be brought about by striking its tendon, mechanical traction, or by the contraction of its antagonist. Thus the curve of the action of the biceps by a single stimulus is not a single curve, but a double one, the second elevation being caused by the reflex contraction due to the action of the antagonist triceps. If the traction applied to a muscle is persistent, with variations in its intensity, such as may be induced by traction on a band of caoutchouc, a series of reflex contractions are induced in the muscle, rhythmical in character and almost constant for each muscular group (10 to 12 per second). Fatigue does not alter the number, but affects the amplitude of the oscillations.

A muscle in a state of contracture acts like the elastic band of varying tension, in relation to the antagonist muscle. Set in action by volitional impulse, by electric stimulation, or by the weight of the limb, it excites rhythmical oscillations in its antagonist which constitute tremor.

Senile tremor and paralysis agitans are explained by the presence of muscles in a state of contracture. The weight of the limb itself

may keep up constant trembling, and the authors have found that spontaneous tremors can be checked, or greatly diminished, by immersion of the patient in a bath, which has the effect of removing the weight of the limbs.

Bourneville and d'Olier on the Pathology of Epileptic Dementia.—Bourneville and d'Olier, assisted in their microscopical researches by Brissaud, give the clinical history and post-mortem appearances in a number of cases of epileptic dementia observed at the Bicêtre. (*Archives de Neurologie*, October 1880.)

In a large number of cases there are no special morbid appearances discoverable post-mortem.

In others again there are well-marked lesions, of the general character of diffuse encephalitis. The seat of these lesions is almost exclusively in the frontal lobes. Their situation here accounts for the absence of paralysis, and the dementia observable.

The lesions are somewhat similar to those found in general paralysis, but they differ in certain points. No miliary aneurisms are discoverable, and, while in general paralysis of the classic type the lesions affect the superficial layers of the cortex, in epileptic dementia, the lesions affect more particularly the deeper layers and adjacent medullary fibres, which appear yellowish to the naked eye. The symptoms which characterise this epileptic dementia differ also considerably from those of general paralysis. It is only in the later stages that the two resemble each other.

There are no extravagant notions, or changes of character, &c., nor inequality of the pupils such as are seen in the early stages of general paralysis. It is a form of dementia intimately connected with epilepsy. The patients differ completely from those, a few in number, who in addition to epilepsy become affected with general paralysis. In such cases the general paralysis exhibits its usual characters. Two cases of this kind under observation establish the fact beyond all doubt.

D. Ferrier.

Rosenbach on Reflexes in Sleep. By Dr. Ottomar Rosenbach. *Zeitschrift f. Klinische Medicin*, 1881. Bd. I. Hft. 2. —Dr. Rosenbach remarks that the various reflexes have not been hitherto systematically tested during sleep. We admit in a general way that the sleeping organism is, as regards its reflex activities, in the condition of a decapitated animal, but, says he, the condition is not quite so simple, and varies greatly

with the depth of sleep, of which we have no exact standard. It is in fact by the reflexes themselves that we are to estimate the depth of sleep, as that of coma. On children, as being the most suitable subjects, he systematically observed the reflexes excited by tickling the palm of the hand, the sole of the foot, and occasionally other parts of the body, also the reaction of the pupil, the cremasteric reflex, the abdominal reflex, and the tendon reflex. He states that at bedtime, or when children are sleepy, the reflex excitability is increased, until they suddenly "set" in the position they happen to be in—a stage of restlessness with which he considers the convulsive stage of chloroform narcosis to be comparable. He says that the first period of sleep, which is light, is characterised by weakening of the reflexes and commencing contraction of the pupil; that in the second or deeper period, the abdominal and cremasteric reflexes and the tendon reflex are abolished; that in the third period or deepest sleep, tickling of the sole of the foot, of the nose and of the ear still liberates reflexes. To the question whether the activity of the sphincters in sleep and in coma is effected by a direct cerebral condition of excitation, or whether it is reflex, he opines that probably there is an augmented cerebral activity protective of the outlets of the sleeping organism. He argues that if sleep were but a cerebral paresis, we ought to get enhancement of spinal reflexes in sleeping, as in decapitated animals; whereas we see that the reflexes, especially the abdominal reflex, weaken as sleep deepens, and that later, various irritations are capable of eliciting precise reflexes. Hence the supposition that not only is there a temporary emancipation of the cord, but at the same time some kind of active restraint of the reflex activities. The author remarks that we may suppose this influence, acting presumably along motor paths, to be strongly exercised in disease, in proportion to its suddenness; thus it should be strongest in traumatic and in experimental paraplegiæ, while slowly acting lesions should bring out the opposite or reflex enhancing influence.

A. Waller.

A Definition of Insanity. Dr. G. M. Beard.—Dr. Beard, in a paper read before the New York Medico-Legal Society, gives the following definition of insanity :—

Insanity is a disease of the brain, in which mental co-ordination is seriously impaired. He claims for this definition that it is a

short description, including all real cases of insanity, and excluding all those that are not insane. Dr. Hammond objected to the statement that insanity is a disease of the brain. To this Dr. Beard replies that by the expression he means that insanity is one of the results or manifestations of disease of the brain. By the use of the word "seriously" he excludes the lighter forms of trance, the temporary and slighter influence of alcohol or opium, &c. If these be prolonged and severe (e.g., the delirium of fever), he would regard the person so affected as insane. Dr. Beard looks to the future to decide the question of the legal responsibility of cases of alcoholic trance.

With regard to the meaning of mental "co-ordination," he is unable to define the word; but believes that "all persons sufficiently intelligent to discuss the subject would agree in their definition could they frame their ideas in language."

The word "responsibility" stood in the place of "co-ordination" in the first form. Dr. Beard gave his definition, and he would still use the former phrase in a court of law.

To meet the objections that naturally arise in regard to the vague and indefinable terms in which his definition is couched, he replies that insanity is a vague condition, a disease of gradations; that only an approximate statement of its nature is possible; and that this statement will be relative to our knowledge of the subject.[1]

Dr. Beard concludes with the following propositions as the outcome of the Gosling case, in which the patient was declared to be labouring under general paresis by all the specialists on insanity who testified in the case, but was allowed to go free by the Court, two general practitioners having testified to his sanity.

First. It showed the need of a definition of insanity.

Second. It illustrated the difficulty of the diagnosis of insanity, the need of repeated visits, so as to examine his behaviour in what may be a remission of the disease.

Third. The interest attaching to the predisposing and exciting causes of insanity.

[1] The first step in accepting a definition is to form a rational conception of what it means and to translate it into other forms of expression. But Dr. Beard admits the impossibility of doing this, and relies on his hearers interpreting the meaning, though all are unable to express themselves in words. It would be of little use then to define insanity in this manner in a court of law, where an immediate explanation of the scientific term "co-ordination" would be demanded. Is this phrase more descriptive than "mental adjustment" or "personal equation," or indeed, is it equivalent to saying more than "insanity is insanity"?

Fourth. The need of distinguishing between the value of the testimony of experts and non-experts.

Fifth. The diagnosis of insanity should be purely psychological, and not at all physical, by contrasting the mental manifestations of the individual in health with the mental manifestations of the same individual in disease, and also with the mental manifestations of the average man in the same condition of life. If a diagnosis of insanity cannot be made out by the mental manifestations alone, it cannot be made out at all.

Sixth. A clear and scientific confession of ignorance should be made, if necessary.

Seventh. Experts should be called and paid by the Court, and there should be a general reform of lunacy jurisprudence.

[It is evident that the fourth, fifth, sixth, and seventh of the foregoing propositions deal with the legal rather than the scientific aspects of the disease. In another place Dr. Beard says, "We may be justified in condemning a man scientifically, when we have no right to condemn him legally;" and again, "The law makes inquiry only of legal irresponsibility as the result of mental incoordination."]

A. R. Urquhart, M.D.

Tassi on a successful Case of Trephining.—Dr. Eugenio Tassi was more successful with his patient. R. P., a wandering merchant, fell asleep upon a wall and lost his equilibrium, striking a pebble on the ground with the left side of the head. He was found unconscious, and was brought to the hospital, when it was found that he had a contusion of the scalp and a fracture at the posterior part of the left frontal bone, with symptoms of concussion of the brain. Consciousness returned; the wound did well for eight days, and there was no constitutional inflammatory action, when one morning it was found that he was paralysed in the right arm and leg, and in a comatose condition. It was determined to use the trephine, which was done thirty-two days after the injury. It was applied "in correspondence with the coronal point (punto stefanico) between the pteric and the bregmatic points." The pia mater was intact; but several detached fragments of bone were found pressing upon it, which were cautiously removed. While this was being done the arm was noticed to move a little. In a few days the mental powers were completely re-established, speech returned, and the limbs recovered their lost activity. During the treatment

there was a spontaneous luxation of the right thigh, the cause of which could not be ascertained. The patient left the hospital completely cured, though he had to use a crutch, owing to the dislocation.

Seppilli on a Case of Atrophy of the Cerebellum.—In the *Rivista Sperimentale di Freniatria* (Anno V., fascicolo iv., 1880) there is a long article on this rare disease by Dr. Seppilli. The patient, a woman of feeble intelligence, had at the age of twenty-eight been afflicted with typhoid fever. During her convalescence there was observed a general tremor and disorder in all the motions of the body. This was accompanied by distinct disturbance of mental power, incoherence of thought and a proneness to weeping and laughing, with hallucinations of sight and hearing. She thought she saw beasts, which terrified her, and heard voices calling to her. This mental disturbance and disorder of motion increased till she was brought into the asylum of Reggio Emilia.

On entering it was found that all the muscles of the face, eyes, and tongue were disordered with clonic contractions. The muscles of the neck were rigid, the arms bent and contracted, and were extended with difficulty. There were occasionally rapid ataxic movements of the arms with contortions of the fingers. The legs were semiflexed and slightly contracted. The general sensibility seemed increased, and tendon reflex of the patella was easily brought out. These disorderly motions increased with the mental excitement, declined with mental calm, and ceased during sleep. A period of excitement alternated with a period of quiet which passed into stupor. The articulation was much hindered by the motor disorders, which were greater at one time than at another. The patient gradually wasted away, and died nineteen days after admission.

The principal abnormality found was the smallness of the cerebellum, which only weighed 56 grammes, less than half the usual weight in women. It was regular in its shape, but hardened in texture, and the grey matter was much wasted. The pons, crura and medulla oblongata were of the normal size. The convolutions of the cerebellum were small and unequal. In the texture of the organ the cells of Purkinje were much less numerous than usual, and those which were found had only half the usual size, and their processes were small and thin. In the median lobe the cells of Purkinje were more abundant, though not so frequent as in a

normal cerebellum. The grey matter of the nucleus dentatus was much diminished. The connective tissue was in excess, and the walls of the meningeal vessels thickened.

The general result might be said to be atrophy with sclerosis limited in great measure to the lateral lobes.

He notices the observation of Stefani, that in pigeons the cellules of Purkinje are found to be atrophied after lesions of the semicircular canals.

Dr. Sepilli cites twelve cases of atrophy of the cerebellum. These do not include the two recently described by Dr. Donald Fraser in the 'Glasgow Medical Journal' for March, which were probably published after Seppilli's paper was written. His observations certainly confirm the prevalent view that the cerebellum has something to do with the co-ordination of the different muscular adaptations necessary to maintain the equilibrium and harmony of the bodily movements. In the cases of Combette, Verdelli, Otto, and Lallement, there was no deficiency of co-ordinating power. In the latter case the atrophy was confined to one side of the cerebellum, and the median lobe remained unaffected, and according to Nothnagel it is this median lobe whose affection leads to the disturbance of co-ordinating power; but in Otto's case the cerebellum was very small, though apparently of healthy texture. Sepilli observes that in four cases of atrophy of the cerebellum (those of Combette, Fidler, Verdelli, and Otto) the intelligence was limited. In the two cases of Dr. Fraser the intelligence was not affected.

It is clear that there are still contradictions in our observations upon disease or deficiency of the cerebellum which cannot at present be harmonised. We have not yet reached a generalisation which will at once explain the function of the organ and enable us to understand the symptoms described as following upon diseased action in its texture.

Riva on Decolorisation of the Choroid in the Insane.—Dr. G. Riva (*Rivista Sperimentale di Freniatria*, Anno V. fascicolo iv.) claims to have discovered a true and special morbid process in the insane. This seems to consist in a diminution of the amount of pigment in the choroid coat, so that through the ophthalmoscope the papillæ appear paler than usual.

Dr. Riva found this condition present in thirty cases out of 117 lunatics studied by him. It seems especially common in pellagrous insanity. Lombroso, in his work on Pellagra, treated it as a phy-

siological process, the result of the depigmentation attendant on the coming on of old age. Riva regards it, at least in the degree observed by him, as peculiar to insanity. He sums up as follows:—The posterior chamber of the eye presents a characteristic appearance in insanity, especially in its intermittent and paroxysmal forms, and where it is dependent upon the pellagrous cachexy. This appearance is caused by diminution in the amount of pigment in the choroid coat and the epithelial pigment of the retina (*epitelio pigmentato retinico*), so that the retina becomes discoloured and turbid.

2. This is not a physiological phenomenon, since it is never observed in so prominent a degree in the sane.

3. The coincidence of these alterations in the fundus of the eye with morbid changes in the brain liable to produce acvtie or passive congestion in the vessels of the choroid, leads us to admit that hyperæmia, which in other parts of the body gives rise to hardening of the tissues, is the most frequent cause of the appearance described.

W. W. IRELAND.

// BRAIN.

JULY, 1881.

Original Articles.

ON INSOMNIA AND OTHER TROUBLES CONNECTED WITH SLEEP IN PERSONS OF GOUTY DISPOSITION.

BY DYCE DUCKWORTH, M.D., F.R.C.P.

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THE sleeplessness which affects persons who are goutily disposed has not, so far as I know, received the attention which it demands. The subject has not been systematically treated by any author with which I am acquainted. Stray allusions have been made to it. There is perhaps not very much to be said about it, but it is fitting that such knowledge as is possessed should be carefully collated and recorded.

In this communication I propose to set forth all that I know about the matter, merely prefacing my observations with the remark that in this endeavour I shall strive to treat the subject from a simple clinical standpoint.

In an essay published in 'BRAIN' in April 1880, I alluded to the fact that there was a "well-marked variety of insomnia" in gouty persons. That this form of sleeplessness is well-marked, and is likewise peculiar, I shall try to show.

In the first place, it may be remarked, that I do not allude to the interruption of sleep which is caused by the sudden onset of an acute attack of gouty inflammation, and which so commonly occurs in the early morning hours. This is an extreme, and very exquisite, instance; albeit, the special phenomena associated with such an attack do indeed, I believe,

afford a type upon which the less-marked and minor symptoms of gouty insomnia are founded.

The simplest form of insomnia which occurs in persons who are goutily disposed is due in most instances, I believe, to dyspepsia. The earliest and best account of this with which I am acquainted, was given by Cullen,[1] who remarked that "persons who labour under a weakness of the stomach, as I have done for a great number of years past, know that certain foods, without their being conscious of it, prevent their sleeping. So, I have been awaked a hundred times at two o'clock in the morning, when I did not feel any particular impression; but I knew that I had been awakened by an irregular operation in that organ, and I have then recollected what I took at dinner which was the cause of it." This sleeplessness is often caused by some particular article of diet which the sufferer digests imperfectly, or may be due to excess of wine, or mixing of various liquors. Very often fatty or saccharine matters in excess, or mixture of fruit and wine, may cause this dyspepsia. There may be no overt symptoms of dyspepsia, but a simple excess in eating, or a single article of diet which is digested with difficulty, may so disturb the cerebral circulation that sleep is interrupted and suspended for a time. I described these symptoms in a paper which was published in 1873, and stated my belief that they were more common in persons who had tendency to gout.[2]

The dyspepsia probably arises from faults not only in the stomach, but in the duodenum and upper part of the small intestine.

Dr. Murchison has described this form of sleeplessness, and attributes it to the hepatic derangement which induces lithæmia and other symptoms of gout.[3] A very noteworthy point about such cases is the particular time at which the insomnia begins. The sufferer retires to rest feeling quite well, and free from any discomfort. But his sleep is rudely interrupted, it may

[1] Works, edited by Thomson, vol. i. p. 127. Edinb. 1827. Cullen did not, himself, connect this symptom with gouty tendency.

[2] On certain forms of sleeplessness. Brit. Med. Journal, Dec. 27, 1873, and republished by Longmans, 1874.

[3] Lect. on Dis. of Liver; edit. 2, p. 590. Lond. 1877.

be by some unpleasant dream, and he is at once aware of uneasiness in the stomach, has heartburn, or flatulence, and perhaps nausea.

If nothing be done, the patient will lie awake, with throbbing head and active flow of thoughts, for an hour or two, when sleep will return. On rising the next morning, he will probably experience some headache, and find his appetite diminished. An attack of hemicrania may perhaps render the next day miserable for him.

Relief may, in general, be readily found by taking a well-diluted dose of some alkaline salt, or by means of a drachm of compound rhubarb powder. If vomiting can be induced, great relief will follow, and a copious draught of cold water may be taken. Sleep will soon return, and no discomfort will be experienced the following day.

Such a form of dyspepsia is plainly a manifestation of a gouty tendency. This incapacity to digest certain definite articles of food is very marked in the gouty, and it is not unfrequently one of the earliest tokens of the disorder.

In youth there may be vigorous digestion for all kinds of food, but as the third decade is approached, the inability declares itself.[1]

The prevention of such disturbance is naturally secured by attention to diet, and omitting each article of food which is found to disagree.

It might be objected that there is nothing very remarkable about such symptoms, and that they are common enough. The characteristic part of the disturbance is the special digestive inadequacy at a very definite period. If we suppose that this enfeeblement is due to the taking of a full meal late in the day, and that the digestive powers would be adequate to dispose comfortably of the same if taken early, which is fairly conceivable, we have no means of knowing whether sleep, were it sought after some hours, would be interrupted under such circumstances. The fact remains that in persons of gouty constitution, sleep is apt to be disturbed by the

[1] This subject has been discussed by me in (1) "Studies of some Irregular Manifestations of Gout." St. Barth. Hosp. Reports, vol. xv. p. 105; and (2) "On Unequivocal Gouty Disease." St. Barth. Hosp. Reports, vol. xvi. p. 185.

irritation arising from their peculiar digestive incapacity, and at a definite period of that process. An interval of four or five hours occurs between the meal and the awaking, the patient being disturbed within about two hours of retiring to bed. This is the time at which attacks of gout are especially liable to come on, and the sufferer is suddenly awaked with pain in the affected part. In this case, as usually in that of gouty dyspeptic insomnia, the patient has retired to bed feeling comfortable, and in his ordinary health.

Other forms of gouty trouble manifest themselves not uncommonly in the early hours of the morning. Thus, attacks of bronchitis, with asthmatic dyspnœa, sometimes replace, or alternate with, regular onsets of gout, and the paroxysms of asthma are very prone to begin, and to disturb the patient, after midnight.

Not only is sleep thus interrupted, but other peculiar symptoms are met with in those of gouty proclivity in connection with the sleeping hours. It has been observed that in cases where a regular attack is expected, but does not supervene, sleep is abruptly broken some hours before the usual time of waking, and does not return. Some horrible dream may lead to this, and the same occurrence may take place for several mornings in succession. Scudamore relates two cases where sleep was merely disturbed by uneasy dreams, and gout was established in the joints on awaking in the morning.

Startings and shouting have been noted, associated, or not, with the dyspepsia preceding or accompanying gout. Severe and troublesome priapism in the night sometimes greatly annoys the gouty, commonly without erotic feelings. Grinding of the teeth during sleep is a symptom met with in those who are gouty. Dr. Graves first called attention to this. Dr. Garrod has no experience of it. I have collected several examples of it, and Dr. Donkin likewise directed attention to some well-marked cases which occurred in the family of parents who were both gouty.[1] In this family there was also history of somnambulism. The father, a gouty man, was a habitual somnambulist in early life, and occasionally in later years

[1] 'Brit. Med. Journal,' Feb. 21, 1880, p. 279.

walked about in his sleep. The mother ground her teeth at night for many years. She was of gouty parentage, as already stated, but had had no overt gout herself. The whole family, of eight children, ground their teeth almost incessantly at night. Most of them were extremely "nervous," and walked in their sleep. They also talked during sleep.

I have record of one case in which there is both tooth-grinding and occasional somnambulism, the mother and maternal grandfather being distinctly gouty. Nightmare and startings of the limbs have been observed with some frequency in persons goutily disposed.

In connection with the subject of insomnia in the gouty, may be noted the fact that many of the special determinations of the malady take place during the night, whether the sleep be disturbed or not. The patient retires to bed feeling in his usual health, but on awaking in the morning he discovers at once some new phase of his malady; it may be muscular pain or stiffness, angina of the fauces, the beginning of a hemicrania, or more or less severe pain in some joint or adjacent texture, such as a stiff-neck, lumbago, or a burning phalangeal joint. These troubles, or some of them, have come on in the night, but have not been sufficient to disturb sleep. Cramps in the calves of the legs are especially prone to vex gouty persons at night, and sometimes precede a severe attack for several nights.

The fact that not only acute attacks of gout are apt to supervene during the hours allotted to sleep, but that other less severe gouty manifestations likewise occur during the night, or are found to have come on at that period, is one amongst many which may be appealed to in proof of the neurotic theory of this malady, for it has this peculiarity in common with several other morbid affections which are conceded on all hands to be distinct neuroses. Thus, epilepsy, neuralgia, spasmodic asthma, gastralgia, angina pectoris, laryngismus stridulus, and hemicrania are all prone to disturb sufferers during the early hours of sleep, or immediately on awaking. In all these cases we have to seek for a cause which determines these outbreaks with such marked constancy in connection with the sleeping state.

From the nature of the case, we have but scant knowledge of most of the physiological conditions which occur during sleep. It is, however, known that the bodily temperature falls both in health, and in most, if not in all, morbid states, between the hours of midnight and six o'clock in the morning. Some observers have noted the minimum temperature to occur between 11 P.M. and 3 A.M., and although the fall does not amount to more than one or one and a half degrees, it has nevertheless a distinct significance as indicating some direct nervous influence on heat-production. Certainly, this constant and normal reduction of temperature is independent of removal of clothing, and abstraction of heat by bedclothes. Again, the subjective sensation of chilliness about three o'clock in the morning is familiar to all who sit up at night, and at that period, too, there is a maximum of weariness and exhaustion, and the greatest instinctive demand for sleep. Even the worst sleepers will commonly fall asleep at this hour, although they may have been miserably wakeful and restless previously.

There is also a greater susceptibility to cold at night-time. The "middle watch" is the most trying in all respects. This is the period of the greatest exhaustion of the whole nervous system, the automatic cerebral activity ceases, and sleep is "the diastole of the cerebral beat."[1] As the old writers put it, the "brain-power is lowered" in sleep.

Digestion is feebler during sleep than in the waking state, and so, too, is the action of the heart, and both the circulatory and respiratory acts are reduced in force and frequency. With the exception of the cutaneous functions, perhaps, all others are at rest as far as possible.

Sleep is more profound in the earlier hours of night, and gradually becomes less so towards morning.

It seems impossible, in view of the foregoing considerations, not to find some reasons for the marked tendency towards irregular outbreak of nervous energy during the hours when so many cyclical processes are modified or interrupted.

In perfect health, and in persons not neurotically disposed, no irregular effects ensue, but in morbid states, and in the neuroses, the hours of sleep are particularly those in which we

[1] Foster, 'Text Book of Physiology,' p. 573.

might expect some outbreak or irregularity, and, as a matter of experience, we find such to be the case in marked degree.

The anæmia of the brain in sleep may have some influence in determining some of these disorders; but this condition is not believed to be the cause of sleep, but only an effect or concomitant of it. In the cases of dyspepsia already considered, there is a manifest source of irritation at a distance; but the peculiarity here is that it only becomes potent at a definite time to disturb sleep, either by the generation of some special morbid product in the course of digestion, which may act from a distance reflexly, or may enter the circulation and rouse the higher centres. These centres may, during their temporary depression, due to sleep, be more than at other periods specially irritable, and in the cases of such persons as are neurotically disposed, they are almost certainly in less stable condition than are those of the healthy.

The direct influence of an excess of uric acid circulating in the blood can hardly be lost sight of in connection with gouty insomnia. It is well recognised that such excess is frequently present without the induction of any overt disturbance, nervous or otherwise, in persons who have no gouty proclivity; but in any case of true gouty habit we must not ignore, as it is perhaps now too much the fashion to do, the influence of what, when in excess, is a real poison in the circulation.

The special determinations of gout to certain parts are inseparably connected with excess of uric acid in such parts, and thus we may fairly conceive that some of the nervous symptoms which occur in the gouty owe their cause to irritation of nervous tissue by this peccant matter. Insomnia may well be one symptom due to this irritation.

It is to be noted that these troubles in connection with the sleeping state are not only met with when the subjects of them are in a very gouty condition, or as precursors of overt outbursts of gout in the arthritic form; they form part of the many minor affections to which persons goutily disposed are sometimes liable. Gout, like other maladies, has varying significance in the particular individual affected, and the fact of goutiness so far modifies the constitution or bodily habit of the patient. Thus, many sufferers have no troubles connected

with the sleeping state, just as many have no urinary difficulties, no hemicrania, and no tophi. Many attacks of gout set in violently by day, and not by night. These facts in no degree minimise the value and importance of accurate observations on the symptoms presented during sleep in persons disposed to gout. We must not fail to recognise their special significance when we meet with them.

Many sufferers are good sleepers in the intervals between severe attacks, and many others can secure good nights with due precautions as to diet and other habits. The particular insomnia described is rather the indication of the gouty habit, than a particular phase of either acute or chronic gout, as commonly understood, and, as such, its importance has not hitherto been clearly signalised. Its recognition is necessary for the employment of the only line of treatment that can truly avail to avert it, and to break the persistence of the habit on which it depends.

Hypnotic agents constitute improper means of relief in such cases as I have described. The gouty taint must be combated. Strict attention to diet, a free dilution of the blood with bland fluids, regulated exercise of both mental and bodily faculties, together with occasional mercurial purgatives, will commonly avail to overcome the misdirected tendencies, and to secure good nights for sufferers from these unpleasant affections.

ON ALLOCHIRIA.

A PECULIAR SENSORY DISORDER.

BY H. OBERSTEINER.

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THOUGH great progress has been made of late in the methods of investigation of diseases of the nervous system, yet there is an affection of sensibility which has hitherto escaped special notice. It consists in the fact that though the sensibility is retained more or less completely, yet the patient is not clear, or is frequently, if not constantly, in error, as to which side of the body has been touched. I would term the phenomenon "Sensory Allochiria," or shortly "Allochiria" (ἄλλος, χείρ) or confusion of sides. This symptom is so easy to determine that no long description of method is required. When we test the sensibility of a patient, on the leg for example, and find that the two points of the æsthesiometer are recognised as such at a moderate distance apart, or when a prick or pressure is fairly localised, and yet, as frequently occurs, the irritation is referred to the other side, we have the condition before us. The power of localisation is retained as to details, while doubt or error exists as to the side touched, the irritation being commonly referred to the corresponding part of the other limb. I believe that the phenomenon is less rare than overlooked, as errors on the part of the patient are apt to be regarded as merely a *lapsus verbi*. In the case of intelligent patients, however, and with due care, it soon becomes apparent that this is not the true explanation. First let me give a few illustrations.

CASE I.—A chemist, ætat. 35, who had had syphilis, and had been exposed to extreme cold, began soon after to suffer with the symptoms of slowly progressive sclerosis of the

posterior columns. After eight years he fell into a state of profound melancholy which lasted for a year. On his recovery from this condition I tested his sensibility. At this time he was able to walk a few steps alone, with his eyes open. Only the essential points need be mentioned.

The patient had a good, if not completely normal, judgment as to the position of his lower extremities, which were freely movable. Heat and cold were readily distinguished in the feet. Simple touch or gentle pressure were not perceived. Investigation with the æsthesiometer showed on the right indistinct diminution of the power of localisation, but not to any marked extent. A double impression was distinctly perceived at the following distances:—

Ball of the toe	R = 20mm	..	L = 12mm
Under the little toe	R = 17mm	..	L = 12mm
Middle of the sole	R = 9mm	..	L = 9mm
Dorsal surface	R = 12mm	..	L = 8mm
Inner side of the leg	R = 10mm	..	L = 7mm

The points required to be pressed deeply in order to elicit distinct sensation. The sensation was markedly retarded. As a rule the point touched was correctly given, only in a few instances an error was made, the patient believing the sole was the part touched, whereas the points of the æsthesiometer were on the toes. Frequently the impression was referred to the other foot, but localised in the correct position, to the astonishment of the patient when he was informed of the error. The patient continued under my observation six months after recovery of his mental equilibrium. He died three years after.

Case II.—W., a labourer, on March 25, 1878, received a severe blow on the back from a beam and was knocked down, paraplegic both as to sensation and motion, but without loss of consciousness. I saw him in December 1878. At that time he had some power of movement of the thighs, but was completely paralysed in the legs and feet. As regards sensibility, the patient made no complaint. He could distinguish hot and cold. On the inner side of the plantar aspect he could distinguish two points at a distance of 9–10 mm.

But he was frequently wrong as to the exact locality of the impressions in the limb, and was seldom right as to the side actually touched. The leg and foot were swollen, the feet turned inwards. On the sacrum there was a slight bedsore; previously the heels had been similarly affected. The urine was not passed involuntarily, but could not be retained long. As to the later progress of the case, the following notes were supplied me by my friend Dr. Pfleger.

During the month of June 1879, considerable œdema occurred in the lower extremities, and sores on the toes. The patient complained of formication in both legs, and soon after febrile symptoms occurred, with erysipelatous redness and swelling of the legs, with considerable diminution of sensibility. These symptoms subsided in a few days, and the sensibility reached its former grade. In October 1879, there were spasms in the thighs, and pains shooting from the hips to the knees. Later the decubitus in the sacral region extended and deepened, and the patient died in a state of marasmus on February 5, 1880. The autopsy was performed by Dr. Weichselbaum.

The body was of medium build, muscular; the lower extremities were œdematous. There was a large decubitus on the sacrum, the size of a small plate. The spinous process of the third lumbar vertebra projected abnormally, and the bodies of the first, second, and third lumbar vertebræ were anchylosed. The anchylosed region was hollow anteriorly, projecting with its convexity into the vertebral canal. The intervertebral discs were only partially retained in this region. The inner surface of the dura mater in the region of the *conus medullaris* and upper extremity of the *cauda equina* was covered here and there with a soft, rusty-brown pseudo-membrane, and united at these spots firmly to the arachnoid. The posterior surface of the *conus medullaris* was flattened, the substance of the cord itself appeared for an extent of 2 cm. of a yellow colour, shining through the arachnoid and pia mater. The lumbar enlargement was not distinct. On transverse section it was seen that in the region of the *conus medullaris* the substance of the cord, with the exception of the anterior columns, and the anterior position of the lateral columns, was converted into

a soft, yellow-brownish mass, strewed with whitish points and lines.

Higher upwards, towards the medulla oblongata, the median portions of the posterior columns was converted into a yellow-brownish wedge-shaped strand, traversed by a few whitish lines. The examination of the cord, hardened in bichromate of potash, was made six months after.

The substance of the cord, from the *filum terminale* to the upper third of the lumbar enlargement, felt spongy and unfit for section. Grey and white matter were undistinguishable. Teased specimens showed that the lowest part of the cord, including the lower third of the lumbar enlargement, was almost entirely degenerated. Besides a finely granular débris there were large numbers of fat granules and nucleated fatty cells. As remnants of the ganglion cells were certain multiform conglomerations, they were without nucleus, but contained one or more vacuoles, and became deeply coloured with carmine. The vessels which traversed the mass were covered on their exterior with a thick layer of fat granules. The connective tissue septa were thickened and contained the mass of detritus in their meshes.

In the upper portion of the lumbar enlargement the anterior horns were distinctly visible, and at the commencement of the dorsal region the whole aspect of the grey matter was beautifully distinct. Fine sections in this region exhibited well-marked ascending degeneration, which occupied the whole of the posterior columns. Only very few normal nerve fibres (9–10) were to be found in the most lateral portions, close to the posterior horns. On both sides also, at the periphery of the lateral columns close to the most external of the anterior root-bundles, there was a region visible to the naked eye by its lighter colour. Whether these symmetrical spots indicated ascending degeneration I have not been able to determine. There was visible also, throughout the whole end up to the medulla oblongata, an increase of the neuroglia, most marked at the periphery. This increase was particularly marked at the points in the lateral columns indicated, but it was not so far advanced as in the posterior columns. These regions were entirely distinct from Flechsig's ascending cerebellar tracts.

In the posterior columns the larger vessels, in consequence of the multitude of fat-granular cells on their adventation, appeared even to the naked eye as whitish cords. Successive transverse sections of the cord from below upwards showed that the degeneration for a length of 3 cm. occupied the whole breadth of the posterior columns, and gradually retreated from the sides towards the middle line, the line of separation from the normal lateral tracts being however at first indistinct. Certainly the degeneration extended quite up to the grey commissure. In the cervical enlargement the degeneration was confined to the columns of Goll, forming a sharply defined wedge-shaped area. The point of the wedge did not however extend as far as the grey commissure. There was no degeneration of the direct cerebellar tracts, owing to the lesion being situated so low down. But in most sections of the cervical enlargement there was a peculiar alteration of the grey substance. On both sides of the cord in front of the head of the posterior horns where they are narrowest, the grey matter was transversely divided by a peculiar, structureless, transparent mass intensely coloured by carmine, and very similar to the mass which is found round the larger vessels in inflammatory processes in the cord (Myelitis, Lyssa, &c.). I do not take upon me to attribute the mass to the same origin, but it seems worthy of note that such an exudation should occur in a region not particularly rich in blood vessels.

In the highest sections of the cord, the ascending degeneration of Goll's columns was beautifully distinct. The ganglion of the *funiculus gracilis* appeared also to have suffered degeneration. Along with fatty granular cells and connective tissue corpuscles, there was a considerable increase in the neuroglia, and the processes of the ganglion cells were fewer than in normal conditions. Yet there were normal fibres, which towards the upper extremity increased in number with the cessation of the degenerative process. Already in the upper cervical regions the degeneration again reached as far forward as the grey commissure, and in the region of the *calamus scriptorius* the sclerotic process seemed to extend forward between the nuclei of the hypoglossal nerves. The degeneration could not be traced further upwards.

Case III.—Dr. N. Weiss kindly showed me a patient who exhibited the symptoms of allochiria, but this was dependent on the nature of the irritation. The case was an ordinary one of sclerosis of the posterior columns. When the patient's foot was touched with the head of a pin, or when the point was pushed in slowly, it happened repeatedly (though the power of localisation was otherwise good) that the patient indicated with the greatest assurance the wrong foot—occasionally also indicating another as the corresponding point. This error was not committed however if the point of the pin was pressed in quickly and forcibly. As my attention was not directed to this point formerly, I am unable to say whether the same existed in the first two cases. But Fischer, to whom I shall refer below, has made some observations on this point.

Case IV.—N. Marie, ætat. 12½, of a neurosal family, some of the members of which have been epileptic. At birth the skull suffered compression by means of the forceps. She had typhus at the age of 3. The girl, who formerly had been very intelligent, became six months ago forgetful, absent, and complained of headache, at first in her forehead, and later in the occiput. She also had abdominal pain without derangement of digestion. These symptoms were regarded by her parents as the prodromata of the menses. At the beginning of February 1880, every evening attacks occurred in which, according to the parents' account, she fell into a state of stupor for one to two hours, during which she spoke unintelligibly, and did not answer questions put to her. Later on she had similar attacks also in the morning. During the intervals she had no recollection of the attacks. I saw the girl first on February 11. I found her in bed during one of her morning attacks. She was almost entirely unaware of her surroundings, playing with various toys, and only with difficulty made to put out her tongue, &c. The face was stupid-looking, and pale. The pupils were square, reacting slowly. There was no motor digest. Pulse 100. She specially delighted in drawing, but drew everything unnaturally and absurd. The attacks then assumed another character. The patient was restless, sang loudly, sprang on the table, &c., and then had muscular

spasms, which occasionally became opisthotonic. During the intervals the following condition was noted. In walking the right foot was dragged. There were spasms in the muscles of the neck and back, and especially when they were talked of; mentally dull, and though she could answer correctly, answered wrongly if admonished. Speech was somewhat like that of a child, but she could articulate well occasionally. She read unintelligibly and counted wrongly. She appeared not to recognise colours, but on strict cross-examination it was clear there was no colour-blindness properly speaking. The ocular muscles did not function altogether harmoniously. The two lower cervical and three upper dorsal vertebræ were tender on percussion. When asked to touch her nose she touched her ear, &c.

As to the cutaneous sensibility, it seemed somewhat diminished over the whole body. Asked to indicate the point where she was pricked in the right leg, she indicated it in the left. After this was repeatedly tried, I directed her attention away to something else, and during this pinched her with increasing severity in her left arm. Only as the pinching became severe she suddenly called out and laid hold of the corresponding part of her right arm.

At the beginning of April, under treatment, the patient had recovered, with the exception of occasional headache and restlessness.

An explanation of this peculiar symptom, allochiria, must be sought for in careful post-mortem examination, and cases like Case II. are desirable in this respect. At present I have not been able to obtain the requisite opportunities.

In the case mentioned we found extensive disorganisation of the lowest portion of the spinal cord. This, however, being transverse myelitis, was not well calculated to indicate clearly the paths of sensory conduction in individual portions of the cord. The complete sclerosis of the whole of the posterior columns for a considerable length above the principal lesion, gives a new proof of the correctness of the views of Woroschiloff, N. Weiss, &c., that the centripetal conduction of the spinal cord can take place without the posterior columns. It

is not however determined by these experiments whether the power of localisation is affected by destruction of the posterior columns or columns of Goll. It must be determined by future research in how far the occurrence of allochiria is related to extensive sclerosis of the posterior columns. As regards the ultimate signification of the small patches of degeneration which crossed the posterior horns I express no definite opinion, as we know so little of the physiology of this region.

The question occurs whether allochiria is an altogether exceptional occurrence in physiological or pathological states. Experience shows that the sensory and motor relations of the two sides of the body are not so independent of each other as is sometimes supposed. We meet rather with phenomena which indicate a close relation between symmetrical regions on both sides. As regards the motor association may be mentioned the well-known phenomenon of associated movements, particularly in paralysed limbs. By forced innervation both sides may be thrown into contraction without the assumption of simultaneous activity of both hemispheres. This bilateral action, so general in the new born, and also visible in certain muscular groups in adults, indicates a preponderance of the lower over the higher centres. Before specially referring to the sensory phenomena, I may mention the similar intimate relationship of the vegetative functions in both sides. As an example of bilateral vegetative function may be taken the activity of the great glands, which has been carefully investigated by Adamkiewicz.[1] Adamkiewicz and others (Adler, Asch, Buccola, Seppili, &c.) have also shown a similar bilateral relationship between corresponding tactile regions of the skin exemplified by the researches on transference of sensibility. The corresponding regions are for the most part antagonistic. If one side is hyperæsthetic, the sensibility of the other side is diminished. The point of importance in the present relation is the bilateral relationship here indicated. This is manifested especially in hysteria, and probably allochiria is occasionally to be found in this condition, as in Case IV. In the other three cases it was associated with organic spinal disease. As

[1] 'Die Secretion des Schweisses, eine bilaterale symmetrische Nervenfunction.' Berlin, 1878.

hysterical hemianæsthesia is similar in its symptoms to central hemianæsthesia of organic origin, so is hysterical like spinal allochiria.

Lately phenomena have been described which indicate not an antagonistic but a parallel relationship between corresponding sensory regions. Dumontpallier[1] has found that ether spray, applied to the left arm, caused also such anæsthesia of the right arm as to allow an abscess to be opened without pain.

I am unable to indicate how often allochiria has been met with in the numerous cases of disease of the spinal cord or of hysteria on record, but the following may be quoted as illustrations.

G. Fischer[2] has recorded two cases of tabes. In the one, in which the symptoms had appeared four years previously after a chill, it is stated that "the localisation of tactile impressions was very inexact. Strong pressure with blunt instruments were constantly referred to the foot not touched. This was repeatedly verified. The sensation of the prick on the great toe was retarded about four seconds. The reflex motion however occurred at the moment of the prick, or was occasionally somewhat retarded, but always occurred long before the sensation of pain," &c. Fischer also refers to another case in which the symptoms had come on a year previously. "Patella reflex very feeble on both sides, but stronger in the left than right. There were regions of tactile anæsthesia. The sensation of tickling was retained, and this gave rise to active reflex movements. Smooth objects held against the sole of the foot were felt as cutting instruments. The patient said also that when the right foot was touched, he also constantly felt something in the corresponding part of the left."

Hertzberg[3] describes a tabetic patient with very marked diminution in the power of localising impressions. "A touch of the finger on the feet could not be distinguished from

[1] 'Soc. Biol.,' Dec. 4, 1880.

[2] 'Zur Symptomatologie des Tabes Dorsalis,' 'Deutsch. Archiv f. klin. Med. Bd. xxvi.

[3] 'Beiträge zur Kenntniss der Sensibilitäts-Störungen bei Tabes Dorsalis. Jena, 1875.

contact of a hard substance, such as a glass rod. Occasionally the patient said he had an indefinite sensation, but could not say whether the right or left leg had been touched, or whether it was the leg or any other part of the body."

Leyden[1] has also observed similar facts. In Case 28 the patient stated he could not perceive light contact with the finger. He felt needle pricks like a touch with blunt instruments. He had very uncertain power of localisation. He even referred a sharp prick with a needle on the left leg or foot, to the corresponding regions of the other limbs.

In another case (Case 27) "the localisation was very uncertain, so that the patient referred an impression on the one thigh to the other." In Case 7 "the sensibility was reduced to a minimum, the strongest pressure was scarcely felt; right and left were frequently confounded."

Leyden, in his 'Klinik der Rückenmarkskrankheiten,' remarks: "The faculty of sensory localisation is frequently so affected in tabes that foot, leg, thigh are confounded, and even when the legs are crossed mistake is made as to the limit touched."

The authors referred to are almost all disposed to attribute this sensory disorder, wholly or almost wholly, to the profoundness of affection of the localising faculty. But the first three cases I have recorded (the fourth may be omitted), as well as Leyden's Case 28, show that allochiria may be manifested when the power of localisation has only suffered to a slight degree, so that, with the exception of the error as to the side, the place touched was quite correctly indicated.

It would therefore be erroneous to look upon allochiria merely as a symptom of great sensory impairment. I am of opinion it must be regarded as a completely distinct symptom by itself. In favour of this also may be adduced the fact that in very many cases of central neurosis allochiria is altogether absent, notwithstanding the presence of a marked degree of anæsthesia.

I do not pretend to be in a position to offer a sufficient anatomical and physiological explanation of this peculiar symptom. But the following considerations may be advanced.

[1] 'Die graue Degeneration der hinteren Rückenmarks-Stränge.' Berlin, 1863.

Apart from the hysterical case, the allochiria occurred all in cases of disease of the spinal cord, mostly in tabes dorsalis. In my second case, which was of a different nature (compression-myelitis), there was, however, a marked degeneration of the posterior columns as shown by the autopsy. It is probable, therefore, as I have already indicated, that the degeneration of the posterior columns is the cause of the allochiria, though we are not yet able to indicate the more minute changes which lead to it.

The above clinical facts also bring out certain physiological points of interest. They show that there is an intimate connection, in a sensory relationship, between corresponding regions of the body, and also that an exactly isolated conduction in the sensory paths of the cord does not exist under all conditions, but under certain circumstances the sensory impulse leaves the accustomed paths and crosses to those of the other side.

THE HEART SYMPTOMS OF CHOREA.

BY OCTAVIUS STURGES, M.D., F.R.C.P.

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It is now generally admitted that there are certain heart symptoms proper to chorea apart from those which are due to its association with acute rheumatism or with anæmia. It is admitted as well that cardiac apex murmur is, more often than was at first believed, the sign of a functional disorder on the part of the valvular apparatus; the precise nature of this defective action has been in great measure elucidated, and it has been repeatedly shown both in chorea and other conditions that such murmurs are temporary and innocuous. With this double advance of knowledge, both in regard to the natural history of the disease in question, and the pathology of the heart, we may consider the cardiac symptoms of chorea from a new standpoint, with a clearer and more just apprehension than heretofore of the facts involved, and fuller and more definite material for their discussion. In what is to follow I propose to investigate the present state of opinion as to the nature and significance of the cardiac phenomena of chorea; to place such opinion side by side with the actual facts of the case; and upon this review, to consider whether by means of any existing hypothesis, or of any modification or combination of hypotheses, the question at issue finds reasonable solution.

The heart symptom of chorea chiefly discussed amongst us is systolic murmur. In regard to the rate and rhythm of the heart, as well as the precise seat of the bruit, there are, as we shall see, doubts and differences; but all observers allow that a soft systolic murmur is apt to arise in the course of chorea, independently of rheumatism or of anæmia, which is variable in tone, disappearing and reappearing, often influenced by

posture and by exercise, most audible in the majority of instances at the left apex, productive of no sensation of its own, and indicated by no outward sign of disturbed circulation.[1] Between this cardiac murmur and the choreic restlessness there is no equality or parallelism. No particular manner or degree of mismovement, and no particular temperament of patient is especially liable to this heart affection. The choreic murmur comes and goes secretly and without notice, there is no recognisable law of its occurrence, and when auscultation has discovered it, neither treatment nor prognosis is greatly influenced by the discovery.

But while these symptoms on the part of the heart, in their physical characters, limited duration and freedom from injurious consequences, remind us most of the so-called functional murmurs, yet have they a near relationship to substantive disease. This is not matter of inference, but of direct observation. There is no fact of chorea better established than this: that in the majority of fatal cases—dying either in the course of chorea or shortly afterwards—a fibrinous bead-like fringe is found edging the mitral valve, and occasionally the aortic valve also.[2] That this condition is the effect rather than the cause of the chorea, we are compelled to assume, in the numerous instances where murmur has gradually developed in the course of the muscular disorder. There are others, however, and especially those which arise out of acute rheumatism (be they few or many), where the sequence is different, and the cardiac murmur, from immediately preceding the chorea, may be taken to indicate an endocarditis which is certainly not the consequence and may possibly be the direct cause of the chorea.

But although endocarditis, upon this showing, seems to be connected with chorea in a double relationship, both as its cause and its product, there are obvious difficulties in the way of admitting it at all. For if endocarditis is really at the origin of chorea, why do the physical signs which should

[1] Such is the general description. Dr. Walshe, however, insists upon the regularity of action, and Dr. George Balfour asserts that the murmur "while it lasts, is constant and unchanging." See Walshe, 'Diseases of Heart,' p. 89; Balfour, 'Diseases of Heart,' p. 166.

[2] 'Statistics of Fatal Chorea.' 'Lancet,' July 17, 1880. 'Pathology of Chorea.' Dickinson, 'Med. Chir. Trans.,' vol. lix.

announce it so often lag behind, and why are they in themselves so equivocal and without the usual accompaniments and results of valve-inflammation? If, on the contrary, endocarditis is not the origin of chorea, but arises out of it, by what sort of agency is this accomplished, and why does not chorea, in virtue of this association, lay the foundation of organic heart disease as acute rheumatism does? Two conflicting doctrines are thus offered for our acceptance, neither of which we can wholly accept, and neither wholly reject. It is certain on the one hand that cases of chorea which happen to die (whether from the severity of the disorder or otherwise) very commonly exhibit a fringing of recent lymph on the mitral valve; it is not less certain on the other that the non-fatal cases (that is of course the vast majority) very rarely exhibit valvular disease in later life. It is certain that both the auscultation signs, and the subsequent history of choreic murmur correspond in most respects with the dynamic murmur of young women; it is not less certain that chorea prefers children rather than young women, and shows after death the actual material of endocarditis.

Such are the difficulties which meet us at the outset in endeavouring to explain the cardiac symptoms of chorea, and it is obvious that the account I have just given in regard to these symptoms does not contain the key to the solution. There still remain, however, other factors of the problem, some which are differently interpreted by different observers, and some which perhaps have not as yet received their due share of notice. Thus, for example, there is the character of the heart's rhythm, which some report to be irregular, and others undisturbed; and there is the further question as to the mode of incidence of cardiac phenomena in chorea in respect of age. It may be that the discrepancies which appear at present in regard to the precise characters and relative frequency of heart disturbance will disappear when we come to compare patients of similar age. We know, for instance, that the liability to such disturbance is in no direct relation to the violence or the particular method of the choreic attack; but we do not know that it has no direct relation to the age of the patient. Again, we know that choreic murmur may occur in-

dependently of any marked change in the cardiac rhythm; but we do not know that this is so at all periods of life, or that disturbed rhythm is not the rule rather than the exception in young children.

In what follows I propose to consider the characters and the frequency of cardiac disturbance in young children as contrasted with those who are older; to compare actual observations upon these points with the more general statements of authors; and, by the help of this comparison and the material it will require, to consider what hypothesis serves best to reconcile phenomena which at first sight seem to be conflicting. I take for this purpose the following:—

1. Dr. Dickinson's seventy-one cases (not 70 as stated) appended to his paper in the fifty-ninth volume of the "Medico-Chirurgical Transactions," and referring to children treated at the Great Ormond Street Hospital.

2. One hundred and thirty-two cases of children treated by myself at the same hospital, but at a late stage of their disorder.

3. Thirty-nine cases of older children and young adults under my charge at the Westminster Hospital.

4. Fifty cases similar to the last recorded by Dr. Owen in the ninth volume of the 'St. George's Hospital Reports.'

These four groups represent respectively, as will be seen more particularly in the sequel—

(1.) Young children from 2 to 12 observed at an early period of their disorder.

(2.) Young children of similar age observed at a later period of their disorder.

(3.) Older children with a few adults.

(4.) Older children, with a considerable proportion of young adults.

In Dr. Dickinson's table the number has to be reduced to 69, on account of two cases with the ages omitted. The children are all young. There is in fact but one, a girl, as old as 13, and but 3 as old as 12; the youngest is 3, the next youngest are 2 of 5; the ages most largely represented are 10 and 11. Taking the 69 cases, with a view to ascertain the incidence of heart affection at different ages, it will be found

that of the 21 children who are 8 years old and under, only 4 are free from heart affection, 3 of these being of the age of 8;[1] while of the 48 children above 8 years old, there are 17 free from heart disorder. If, however, we state the numbers somewhat differently, classing the children of 8 amongst the elder ones, we shall then find that in 11 children under 8, there is but one free from heart affection, while in 58 of 8 years old and over, there are at least 19 free from any abnormality of cardiac action which can fairly be reckoned.[2] As regards irregularity, the 69 cases give 20 such examples, 12 of irregularity alone and 8 of irregularity together with murmur. Of the 21 children 8 years old and under, there are 5 with hearts irregular only, and 3 with irregularity together with murmur. Of the 48 children over 8 years old, there are 7 with hearts irregular only, and 5 with irregularity together with murmur.

I take next a second group of children from the same hospital who were under my own charge, having been received from one or other of my colleagues.[3] The ages are still between 2 and 12, as in the first group, but the children come under observation at a later stage of their disorder; the great majority were chronic cases, some few were convalescent. The total number of these patients was 132 (appearing as 137, from 5 of them having been twice admitted). The age distribution is as follows:—

12 years old are only	2
11 years old are	24
9 and 10 years old	47
8 years old and under, 36 of these being under 8	59
	132

[1] Of the 17 children of 8 and under having heart disturbance as many as 5 had had acute rheumatism, to which therefore the heart condition might be referred. On the other hand, of the 31 children over 8 having heart disturbance, as many as 6 had had previous attacks of chorea, to which period therefore rather than to the age recorded, the heart disturbance might be referred.

[2] I have made these calculations from the tables themselves (to which I refer the reader), and they do not precisely correspond with Dr. Dickinson's own summary (on p. 34 of his paper). This latter, however, has reference not to the *ages* of the patients, but to the several *causes* of the chorea.

[3] I have referred to this series elsewhere, in connection with the rheumatic origin of chorea. 'Lancet,' Nov. 29, 1879. It is not given here *in extenso* out of regard to space.

In the whole 132, 47 (more than a third) have heart murmur or irregularity. The 59 children of 8 and under have 24 examples of heart disorder; the 72 children over 8 have 23 such examples. In other words, the proportion of heart disturbance is about 40 per cent. in the younger children, and about 32 per cent. in the older ones.

Again, if we take the extremes of age in both directions, that is to say, if we compare the 26 children of 11 and 12 (the oldest we have to do with) with 36 who are *under* 8, we have 9 of the former having heart disorder against 16 of the latter, the actual percentage of heart disturbance being thus somewhat greater in the younger than in the older children. Of cardiac irregularity, as apart from murmur, not including mere unevenness of rhythm, there are but 9 examples, 6 of these referring to the younger children.

These cases, therefore, representing the same ages as Dr. Dickinson's, but referring to a later stage of the affection, yield a much diminished proportion of heart disturbance of whatever kind. It is a little over a third, whereas in the former table it is over two-thirds. And what is still more remarkable, irregular action of the heart, largely represented in Dr. Dickinson's list, hardly appears in mine, and is almost confined to the younger patients.

It thus appears probable, taking these two tables as they stand, and comparing them together, first, that the heart affection of chorea is not less but more marked in early than in later childhood; secondly, that observation of the disorder at a late period discovers less heart disturbance than at an earlier, and especially that cardiac irregularity, a distinguishing feature of the chorea of early childhood, is found at the beginning rather than at the end of the attack.

Such, I say, are the conclusions to be drawn from a bare enumeration, and so long as we confine ourselves to children the majority of whom are not over 10, they are conclusions which need no correction. But when we enlarge the view and admit older children and adults; in other words, when we go from children's hospitals to general hospitals, we can no longer accept the mere figures without some attempt at adjustment on account of disparity of age. Thus, for example, all other

things being equal, the greater the age the larger the proportion of heart disturbance independent altogether of chorea. We have to consider besides, that in comparing childhood with adolescence, in respect of the signs we are considering, we have to encounter with the later period those functional murmurs (variously named, and attaching especially to young women) which, although they be altogether distinct from chorea as such, are not in practice distinguishable from it. Add to this that the older the patient the greater the probability that the chorea is not the first, but the second or the third attack, and the heart disturbance therefore but a revival of what has occurred in childhood, according to the well-known rule of the disorder to repeat itself. Such cases illustrate in fact the liability of *early* childhood in respect of heart sympathy: they are taken to illustrate the liability of the more advanced age at which the patient comes under notice. Again, some account must be taken of the previous occurrence of acute rheumatism, which is likely to appear in larger proportion in the histories of the older patients than of the younger, and to contribute its share of heart murmurs which are not choreic but rheumatic. It would be quite unfair, I say, to attempt the comparison we are now about without making allowance for discrepancies of this kind; without providing, that is to say, for the fact that the younger the child the more likely the heart disturbance to be intrinsic, the older the child the more likely to be accidental or contributed. In any exact computation as to the age-incidence of heart disturbance in chorea, it would be necessary of course to exclude from the comparison anæmic and chlorotic girls, as well as those who in earlier childhood had had either chorea or acute rheumatism. In the case of such children as we have been considering, all of them young, the correction necessary on this account would not amount to much, and we need not stay to make it; but in the case of the older patients we are now coming to, it makes a very wide difference. It would be altogether delusive and misleading to place the old and the young side by side with the view to an even comparison in respect of heart sympathy in chorea. Numerical equality between the two classes, or anything near an equality, would

indicate, without doubt, that the heart was more often implicated in the chorea of the young than of the less young.

How the figures stand in these respects with older patients, and how large a correction is needed for such reasons as have been mentioned, will be seen from the examples of chorea which remain to be quoted, viz., the 39 of Westminster Hospital and the 50 of St. George's Hospital. In the 39 Westminster cases 20 are 12 years old or over, 5 of them being young women. In the 50 cases reported by Dr. Owen in the 'St. George's Hospital Reports,' vol. ix., of two following years, as many as 27 are 12 years old and over, 11 of these being young women and 6 young men. We have thus in the four series of cases now rehearsed a constant rise in the ages of the patients. Dr. Dickinson's are the youngest, and the St. George's cases are by much the oldest, the Westminster series standing midway, and my own Children's Hospital cases differing from Dr. Dickinson's more in the duration of attack than in age of subject.

The 39 Westminster cases are distributed as follows:—

13 years and over (2 being young women)	15
12 years old	5
11 years old	5
9 and 10 years old	9
8 years old and under	5
	39

Of the whole number, 13 would have to be excluded, 5 on account of acute rheumatism at some earlier period, and 8 on account of previous attacks of chorea. The remaining 26 are made up of 19 who have no cardiac disorder, and 7 who have either murmur or irregularity. Of the 19 who have their hearts free, 15 are over 11 years old; of the 7 who have cardiac murmur or irregularity, only 1 is over 11.

In the two tables of cases of St. George's Hospital there are 15 examples of heart disturbance in 50 cases, 10 of these 15 are found among the 27 cases over 12 years old; only 5 are found among the 23 cases under that age. Upon this showing, it would seem that cardiac symptoms in chorea are not more rare but much more common after 12 years old than before.

Upon examination, however, of the 10 individuals exhibiting heart murmur and over 12 years old, it appears that 4 of them have had previous attacks of chorea, 1 had pericarditis while in hospital, 1 developed murmur just after acute rheumatism, and 2 were weakly girls, aged 14 and 15 respectively. There are thus 8 of the 10 whose murmurs are accounted for apart from chorea altogether. We get but two properly choreic murmurs in 27 choreic patients of ages ranging from 13 to 18, as large a proportion, I am disposed to think, as will be commonly found at that period.

There is indeed considerable difficulty in fixing the time of life when the liability to heart disturbance in chorea finally ceases. The evidence already adduced shows, I think, that childhood is particularly liable to it, and that irregularity of rhythm distinguishes this earliest liability, whether with or without systolic murmur. It is also generally agreed that the rare chorea of advanced life has little connection with heart disturbance. But of the middle period our knowledge is far from precise. For it so happens that at the very time when, with the progress of development, the frequency of cardiac murmur in chorea is obviously lessening there comes a period of life (and especially with young women) when the precise significance of the symptoms we are discussing is hardly discoverable. Murmur may be choreic or (so-called) anæmic. It would seem impossible at such time to determine with any approach to certainty the proper liability of the heart in connection with chorea. We know only that so soon as this interval of obscurity has passed, and, with the completion of adult life, the indications furnished by the heart are again trustworthy, cardiac sympathy in chorea is hardly recognisable.

From the facts now adduced several conclusions seem to follow. Heart symptoms in chorea occur most frequently during a period which is included between 2 years of age and about 7 or 8. The age of greatest liability to choreic heart disturbance thus partially coincides with the age of greatest liability to chorea. Again, the abrupt decline in the liability to chorea, which occurs at about 15, corresponds with a similar decline—if it be not rather a cessation—of the dispo-

sition of the heart to share in the disorder of the voluntary muscles.

It has been observed already that irregular cardiac action, either alone or preceding or accompanying the murmur, is a pretty frequent feature in the younger children of Dr. Dickinson's table. In my own children of similar age, but in a later stage of chorea, it is much less common. In the Westminster cases, where the patients are older, it is also rare, and in the St. George's cases, older still, irregularity hardly occurs. This particular symptom, in the younger subjects, is often the single sign of the heart's affection. I believe, although I cannot show by tables, that there are very few little children, if there be any, in an early stage of chorea, who do not exhibit in some degree unevenness of cardiac rhythm. Such unevenness will sometimes develope into murmur and sometimes not, murmur which is almost always variable with position, sometimes best heard over the pulmonary cartilage rather than at the apex,[1] apt to come and go, and often finally lost sight of before the chorea itself has disappeared. The second pulmonary sound is seldom (Dr. Walshe says never) either accentuated or doubled.

These statistics in regard to the age-incidence of the heart symptoms of chorea enable us to render a fuller account than that first given of the particular phenomena. Dr. Walshe's assertion that the cardiac action is regular must be limited to the older subjects, or to the later stages of the disorder. Dr. Bristowe's statement that the greater number of choreic patients have some cardiac defect, irregularity amongst the rest, must be qualified by adding that this large proportion refers to children, and that irregularity as a sign of heart disturbance is almost confined to them.

[1] The fact of mitral regurgitation being most audible in this situation, is well explained by Naunyn, as reported by Rosenstein (Ziemssen's 'Cyclopæd.' vol. vi. p. 122.) He points out that the second intercostal space, close to the left edge of the sternum, coincides with the point of the left auricular appendix, which winds round the pulmonary artery and lies in front of it. Now with the abnormal current of blood flowing towards the auricle, we can easily understand, says Rosenstein, "how the sound should be conducted to the spot mentioned better than towards the apex, more especially in cases where the appendix is long enough to lay its point close against the anterior wall of the thorax."

Thus the several factors of the problem with which we have to deal summarily stated are these:—

1. In the course of the chorea of childhood the heart is apt to become irregular or uneven, and its first sound to be followed by apex murmur, which is variable in pitch, influenced by posture, seldom audible in the axilla or at the angle of the scapula, and which disappears along with or shortly after the chorea, the heart and the circulation suffering no injury.

2. This liability on the part of the heart to what, from its signs, would seem to be a functional disturbance is independent of the violence or method of the chorea, but dependent upon the age of the patient, the younger children being the most, and the elder the least liable, while beyond childhood there is little if any liability of the kind.

3. These heart signs of chorea—acute rheumatism being excluded—give rise, as a general rule, to no symptoms whatever affecting the health or comfort of the child. They make no apparent difference to the prospects of recovery or the structural integrity of the heart. Nevertheless, choreic children having this murmur and happening to die, either with or shortly after recovery from chorea, very commonly exhibit a beading of recent lymph on the mitral valve.

Such, I say, are the chief statements which statistics seem to warrant. I will venture to add another, which, so far as I know, has never been statistically reckoned, but which no one will gainsay. It is, indeed, the most constant of all the heart symptoms of chorea, and met with at a later age than the rest. I mean the acceleration of the heart and pulse.[1]

Now there are two hypotheses of the heart phenomena of

[1] Ziemssen says "cases of arhythmia are certainly very rare, and there is hardly anything published about it." On the other hand, he regards acceleration of the action of the heart as common, and acceleration of the pulse as quite constant, although, as he believes (I am convinced erroneously) the bodily heat is not changed even in severe cases. (Ziemssen's 'Cyclopæd.' vol. xiv. art. Chorea, p. 436.)

The heart symptoms mentioned in the text are not the only ones to be met with in chorea. I have heard presystolic murmur, and both accentuation and doubling of the second sound, and this in cases where there was sufficient evidence that the heart disturbance was purely choreic. But I am not able to say at present to what ages these exceptional signs chiefly attach, or what is their proportion to the commoner symptoms.

chorea, each of which has special regard to certain of the facts just mentioned, although neither is able to reckon with them all. The two are, in a sense, mutually antagonistic, inasmuch as what the one rejects the other prefers. Both theories have their supporters in this country. The one asserts that the heart symptoms of chorea are due to endocarditis, the precise relationship of the muscular disorder to the valve inflammation being variously interpreted. The other asserts that the cardiac signs are intrinsic, a part of the chorea itself. That there is some evidence in favour of each of these views we have already seen. The task before us is to reconcile them.

To the belief that the heart murmur of chorea is due to endocarditis I know of nothing in the actual clinical symptoms that can be opposed. Our information in regard to the physical signs of endocarditis *per se* is, indeed,[1] vague enough, while the probability of its occurrence in chorea is directly favoured, as we have seen, by post-mortem evidence. The distinction between organic and functional heart murmurs, founded on hard and fast lines as to the transmission of the bruit, is of doubtful validity. "A well-pronounced functional murmur," says an excellent observer,[2] "may be as diffused and transmitted as an organic." "A systolic apex murmur," says Dr. Bristowe, "is a positive proof of regurgitation." We have indeed, just as much evidence, so far as physical signs are concerned, of endocarditis in the course of chorea as we have of it in many instances of acute rheumatism. In both cases alike a soft and not conveyed murmur may be proved, in the event of death, to depend upon valve deposit, and in both

[1] "There are few diseases," says Rosenstein, "the presence of which is diagnosticated so arbitrarily as that of original acute and subacute endocarditis." He proceeds to point out the necessity of "proving the existence of *other symptoms in addition to the mere murmur*, especially intensified second sound in the pulmonary artery, the localization of the murmur, or transverse hypertrophy of the cardiac volume." Rosenstein, art. Endocarditis, Ziemssen's 'Cyclopæd.,' vol. vi. p. 90. Dr. Bristowe says, 'If in the progress of any of those diseases of which endocarditis is a common complication, we detect a recent cardiac murmur, and *if further observation proves this to be a permanent phenomenon*, we cannot reasonably doubt that endocarditis is present." (Bristowe, 'Theory and Practice of Medicine,' 2nd ed. p. 522.)

[2] Dr. Nixon in 'Dublin Journal of Med. Science,' vol. iv. p. 575.

alike (I do not say to a like extent), when death does not so intervene, the murmur has been found to disappear. In frank recognition of these facts, Dr. Wilks expresses the belief that all mitral systolic murmurs associated with chorea are organic; and Dr. Sansom,[1] in a short summary of the prevailing opinions upon the subject, admirable for its conciseness, arrives at a like conclusion.

But when this much has been granted and endocarditis is admitted, how much are we the better? What is the mode of origin and what is the sequel of such endocarditis? It is but exchanging one difficulty for another. For we have to suppose, upon this hypothesis, not only that endocarditis may arise in and out of chorea, and that the younger the child the greater the probability of this event, but also that cardiac irregularity sometimes precedes and sometimes takes the place of regurgitation, whilst none of the injurious after consequences which attend endocarditis in its other relations are found to ensue here. In face of such grave objections, we are forced to admit that, although physical signs do not contradict it and post-mortem evidence may seem to be directly in its favour, the theory of choreic murmur (not to speak of the other signs) which invokes endocarditis is too difficult. It is inconsistent with all that we know of that inflammation in its various pathological relations; and apart from these we know remarkably little about it.

It is in fact by virtue of this vague knowledge that endocarditis maintains its hold in this connection. A form of heart inflammation special to chorea, and having a history and mode of termination of its own, is a creation of too shadowy a kind to be easily dealt with. But so soon as anything definite and within reach of investigation is alleged in regard to it, when, for instance, it is said (as Trousseau, Bouillaud, Roger, and other French authors say) that the endocarditis of chorea is in fact rheumatic, although the rheumatic element is itself latent, we have a distinct yet still but partially tangible issue. Of the relationship between chorea and rheumatism I do not propose to speak in this place. I have endeavoured to show elsewhere that although real it is rare and exceptional, and

[1] Sansom, 'Diseases of the Heart,' p. 89.

both Walshe and Hayden are of the same opinion. However this may be, no one will refuse to admit that the cardiac symptoms of chorea are observable sometimes when there is no open rheumatism either past or present. Now if in all such instances we are to say, in the first place, rheumatism is actually present, but after a secret and ineffable manner, and in the second place endocarditis is the consequence of that rheumatism, but it is an endocarditis which is exceptional in commencing after the chorea has begun and disappearing along with it, we account for the facts no doubt, but after a fashion which rather begs the question than provides for it.

We are thus compelled to regard the heart symptoms of chorea, taking them in their entirety and not by arbitrary selection—the early irregularity, the variable mitral murmur, the very frequent acceleration of heart and pulse with frequent fluctuations in the rate of both—as being altogether special and peculiar. They are signs of the heart's sympathy with the voluntary muscles, and are seen most at that early time of life when the antecedence of acute rheumatism is the least probable. The heart suffers by whatever mechanism or nervous influence, part and parcel with the rest of the muscular system, or rather it is apt to suffer. Such a conclusion, I say, in some sense or other, seems self-evident, and is indeed involved in the statement that the heart symptoms of chorea are met with in no other association.

Obvious and inevitable as it may be, it is far from being easy of application. For if it be granted, in the words of Dr. Walshe, "that the apex murmur of chorea is plausibly ascribable to disordered action of the muscular apparatus connected with the valve," why does not the same disordered action extend to the ventricular walls? It may be answered (although Dr. Walshe does not admit as much) that in young children there is both disorder of rhythm and regurgitation; but the answer is insufficient. These two signs do not commonly concur nor do they vary together. And it is not young children alone that we have to consider, but the elder patients as well; and in these latter, while murmur is not infrequent, any marked irregularity of cardiac rhythm is

certainly rare. Thus we have to suppose that the fibres of one and the same muscle are acting regularly as regards the ventricular wall, but irregularly as regards the papillary muscles. "We should be forced to admit," says Dr. Hayden, "that whilst the greater portion of the length of certain muscular fibres contracts with perfect order and regularity, the remaining and smaller portion of the same fibres acts spasmodically and out of harmony with the former. No physiologist," he adds, "bearing in mind the unity of nerve centres and the community of nerve distribution enjoyed by both portions of the same fibres, would admit such a doctrine."[1]

There are other objections equally formidable. It is not in the nature of chorea to dissever the normal combinations of muscular movement. It stirs the very same muscles that are employed by purpose or emotion, and moves them in a similar way, only less precisely and with less definition. Apart from anatomical considerations, therefore, a choreic spasm of the heart, which should select the papillary muscles and leave the cardiac walls, would be untrue to the pattern of chorea elsewhere. And again, if this choreic conduct of the heart be but an extension of the same disorder which affects the other muscles (an extension, as Dr. Kirkes points out, to which there is no parallel in any other involuntary muscular organ), the most violent and general chorea ought to exhibit this cardiac disturbance the most. But it is not so. On the contrary, the violent chorea of puberty has seldom either cardiac murmur or cardiac irregularity, while the chorea of the young child, which is but rarely violent and is sometimes rather a paresis than a disorder of movement, exhibits both these forms of heart affection very often.

And even if these difficulties should be overcome or the ground of some of them disputed, it would still, I think, be hard to understand how the great centre of circulation and (in one sense, at all events, within the experience of everybody) of emotion should become the seat of a mismovement at all resembling the choreic contortions of the visible muscles without giving rise to sensations of grave disquiet, sensations, be it remembered, which, so far as young children are con-

[1] 'Diseases of Heart and Aorta,' p. 268.

cerned, the favourite subjects of such cardiac disorder, are conspicuously absent.

It would thus appear that both the hypothesis which supposes endocarditis to be the cause of choreic murmur and the hypothesis which supposes an extension of the muscular infirmity to the heart to be the cause of it are fallacious. Both theories commit us to too much. And if, in this strait, we have recourse to vaguer expressions having regard to the "impoverishment" or "vitiation" of the blood, we neither provide an adequate explanation nor allege a demonstrable cause. But while thus compelled to abandon one position after another, we must beware lest by too precipitate a retreat we quit ground which it is essential to hold. We may even venture so far upon the assured facts of the case as to make some sort of stand and to affirm something. If the views just announced cannot be held in their entirety they must be held with some modification or other. There is no alternative. Part of the perplexity arises, as has been shown, from a consideration of the harmlessness, variability and limited duration of the choreic murmur. But if these facts are in themselves secure we may rely upon them to force at least this conclusion, that the conduct of the heart in chorea is similar to its conduct in anæmia. Whatever explanation, therefore, applies best to the precise mechanism of the so-called functional or dynamic murmur cannot be altogether foreign to the circumstances of chorea.

It is now generally admitted that the murmurs once called hæmic, anæmic, and by similar names implying dependence on some blood change, are much more often than was at first supposed produced by actual regurgitation, due to a temporary defective action on the part of the mitral valve. Such murmurs are to be heard in purpura, in anæmia, in the general muscular relaxation after various nervous strains, in epilepsy, as well as in typhus and enteric fever, and even in simple pyrexia. This dynamic mitral bruit may, by exception, be harsh and far sounding, audible in the axilla and even at the lower angle of the scapula, yet nevertheless by its ultimate disappearance give ample proof that whatever may befal the valve during its continuance (and there is some reason to believe that a material change actually occurs) is not abiding

or permanently mischievous. So much, I think, is generally admitted. The question for us now is, whether the same explanation of the mechanism of this murmur which applies to its association with the conditions just mentioned applies also to its association with chorea.

No one, so far as I know, has more ably discussed this subject than Dr. Nixon.[1] His exposition of the various ways in which dynamic apex murmur may arise, and of "the perfect unity and correspondence in action of the fibres of the walls of the ventricles and of those which are connected with the valves, which is needed in order to ensure complete closure," suggests to him the inquiry whether this nice correspondence of movement might not be so disturbed by altered nutrition or innervation as to render such closure for a while defective. The tremor of the voluntary muscles, which is apt to follow upon their violent exercise, is adduced as being analogous to this disordered cardiac action. Dr. Nixon concludes that functional mitral murmur results from "this want of correspondence between the fibres of the ventricle, which obliterate the cavity, and those which close the valve;" an altered function due, as he believes, "to some defect in the vital power or condition of the heart itself, which leads either to atony of the papillary muscles or derangement in the rhythm of their movement." In the further observations of the same author as to the means of separating this murmur from the organic, he alludes to its changeable character, alteration or even disappearance with the upright posture, and the absence (in most instances) of accentuated or double second sound. Thus, in all its essential features, the murmur which Dr. Nixon describes corresponds with that of chorea; and, except in cases of temporary debility, anæmia and certain conditions already particularised commonest in the female sex, it is seen in no other connection.

Dr. Hayden, assenting in part to these views, would attribute the dynamic murmur not so much to a want of correspondence between muscular fibres, as to atony or parietal debility of the walls of the left ventricle.[2] Weakness and relaxation of the papillary muscles, he admits as a possible cause of mitral regur-

[1] 'Dublin Journal of Medical Science,' vol. lv. p. 572, &c.
[2] Hayden, loc. cit. p. 274.

gitation. Spasm, or irregular action of the same muscles, he does not admit as a possible cause of it. This regurgitant murmur, "caused by atony and partial yielding of the walls of the left ventricle at the acme of systole," Dr. Hayden has long associated with anæmia, purpura, and excessive use of tobacco. His later observations lead him to include chorea and fatty heart in the same category.

But the latest and least reserved expression of the opinion that dynamic murmur is in fact dependent on muscular paresis is that of Immermann.[1] "The muscular tissue of the heart," says he, "owing to the altered state of the blood, is easily fatigued, and this liability to premature fatigue extends to the papillary muscles connected with the auriculo-ventricular valves. After any undue exertion of the cardiac muscle a temporary paresis of the musculi papillares ensues. In consequence of this the valve flaps intrude into the auricles with every ventricular contraction, that is, a transient functional insufficiency of the tricuspid and mitral valves is established. As the organ regains its normal energy these grow fainter and ultimately disappear." He goes on to describe the fatty degeneration of the papillary muscles which sometimes ensues upon simple anæmia, and which explains both the occasional permanence of these murmurs and their occurrence in connection with fatty heart.

It will be admitted, I think, that both the time of appearance and precise manner of the choreic murmur, as well as its mode of origin and duration, varying intensity and limitation to the period of youth, are all nicely provided for in the hypotheses just rehearsed. If only it be admitted that the heart's disorder is really a sympathy with the muscular disturbance, an inexact and precarious correspondence between the ventricular contraction and that of the papillary muscles would seem to serve for chorea even more than for the conditions to which Dr. Nixon applies it. Still better, and without making any assumption as to the heart's sympathy, might we admit the doctrine of partial paresis of the valve apparatus, and insist that the excessive muscular movement of chorea is the precise condition which, upon the hypothesis, should give rise to

[1] Immermann, art. "Anæmia," Ziemssen's 'Cyclopæd.' vol. xvi. p. 399.

murmur such as we hear in token of fatigue on the part of the heart. Adopting either of these views, we might say with Ziemssen that "change in the valvular sound is *à priori* more likely in chorea than in any other affection."[1]

But the matter is not so easily settled. Let it be granted that the choreic murmur is due to a functional disorder on the part of the mitral and probably also of the tricuspid valves, by means of which a temporary and variable regurgitation is permitted (and I think so much of admission is inevitable), we have to ask, in the next place, why this regurgitation, whether it be due to faulty adaptation of the several parts of the heart to one another, or to the paresis of fatigue, is not in harmony with the other symptoms? If the murmur betokens a sympathy on the part of the heart with the disordered action of the other muscles, it ought to appear when chorea is the most generalised; if, on the other hand, it betokens a fatigue paresis, it ought to appear when the chorea is most violent and prolonged. But it does neither. The sympathy of the heart (if it be rightly so called) goes not with the manner or the degree of the chorea, but, as had been shown, with the age of the patient. And, moreover, we should be taking a very partial view of the subject were we to limit the heart symptoms to cardiac murmur. This, as we have seen, is but one, and not even the chief, symptom of choreic heart disturbance. There is besides the unevenness or irregularity which belongs especially to early childhood, and, most constant of all, there is the accelerated pace of the heart. Again, we have not merely to account for the presence of these additional signs, we have to fit them into their places, and to show why the heart, seldom failing to share the muscular disorder in one way or another, exhibits this mode of disturbance or that according to the age of the subject. We may accept either account of the mechanism of dynamic murmur, and without much violence apply it to suit the circumstances of chorea: that is a little gain, no doubt. But when that is done there remain these further symptoms, which, unlike valvular murmur, can compare with nothing to be found in other analogous conditions, unless indeed the cardiac unevenness and its acceleration remind us of what is sometimes met with in

[1] Loc. cit. p. 440.

hypochondriacs and hysterical women, the least promising subjects to throw light upon obscure places in pathology.

It would seem then that to understand the conduct of the heart in chorea we must look to chorea itself and the modifications it exhibits at various ages. Here is an affection which both in absolute frequency and in its tendency to implicate the heart, attaches itself to young children most of all, to older children somewhat less, to adults hardly at all. At each of these periods chorea, both in the limbs and at the heart, shows itself after a particular manner. If, then, the action of the heart in chorea is throughout in real harmony with that of the voluntary muscles, the clue to the secret we are in search of must be sought in the special characteristics of those periods of life to which chorea especially attaches—characteristics, that is to say, in which childhood and youth differ from infancy on the one hand and adult life on the other. If the reader is not already wearied by the length of this exordium and the somewhat circuitous path we have had to pursue, I would venture in such space as remains to take a final survey of the subject from this point of view.

I would observe, in the first place, that the chorea of the post-infantile period is the immediate successor of the muscular spasm or convulsion of the infantile period. At an age varying with the "forwardness" of the child, the one liability takes the place of the other. The very same material cause (as for example, intestinal irritation, or the troubles of dentition) will excite convulsion in infancy, and chorea a little later. Again, the chorea of early childhood will sometimes commence with convulsion, or the two affections may alternate, the same limbs being affected in both cases. Little, of course, can be certainly known as to the actual sensations of the child at this early age, or of the mode in which physical pain gets mentally interpreted; but it depends probably in some measure upon the degree of completeness of such interpretation whether the response shall be by way of convulsion or by way of chorea. However this may be, the general muscular restlessness of the young child which passes for chorea is not only in close union with spasm in the way just mentioned, it often bears a direct resemblance to it in form. There is a uni-

versal unsteadiness in which the muscles of the abdomen and of respiration share as well as the diaphragm and larynx. This generalised chorea which thus succeeds to convulsions is, so to speak, the simplest form of the disorder, without variety so far as the movements of the several parts of the body are concerned, and without any mental admixture. It is an aggravation of the natural unsteadiness of childhood, an universal and equable overmovement or mismovement extending to all those regions which it is the nature of chorea as a motor disorder to affect, and it is this merely. There is no incoordination, for the muscles have not yet learnt to act in concert; there is no distinction between the movement of one part and another, for the several muscular uses are not yet differentiated, and the limbs are stirred indifferently without method or purpose. Such general, featureless agitation of the body is the proper response to a source of irritation which appeals to no one part in particular and has no mental representation.

With maturer age the bodily movements become varied and definite, and each department of the muscular system sets about its separate business. And as with movement so with mismovement. The chorea of later life is a definite deformity which alters and disfigures the natural carriage in this way or that according to its seat. It selects those muscles which are the readiest to respond to emotion, and moves them as they are wont to be moved by the passions and employments of life. Thus the choreic distortion of the face is one thing, of the legs another, of the arms another; and this separateness becomes the more distinct as the individual grows and his individuality, as it is called, becomes more and more marked. The mismovement of chorea betrays the age of its subject, no less than does the natural movement. And as life goes on chorea becomes more and more distinctly localised. In its latest form it is a partial disobedience or misconduct of certain muscles, rendered inveterate by habit, and incorporated as it were with the rest of the bodily movements.

My conjecture is that the conduct of the heart in chorea is in strict accordance with these successive modes or differentiations of the muscular disorder. The heart sympathises with

chorea in one way or another throughout life, or at any rate until that period is reached when the affection is both rare and difficult to identify, and has ceased to apply to the whole system. Not to speak for a moment of the valvular murmur, the heart is unrhythmical or irregular in early childhood,[1] accelerated in youth and left undisturbed in old age. Thus the earliest period of heart sympathy, that of irregularity, with or without acceleration, corresponds with chorea, a purely motor disorder, the earliest transition from infantile convulsion and differing from it mainly in the character of the movement and the implication of the emotional centres; but resembling it in its exciting cause, wide range, and conformity everywhere to a single pattern. In the heart as well as the limbs, in the tongue, the speech, the diaphragm, in all the parts in fact accessible to emotion, yet without any mental representation of emotion, the choreic disturbance of little children is a motor disorder, merely excited very often, like the earlier convulsion, by a material irritation.

In like manner the cardiac acceleration of youthful chorea, with or without murmur, corresponds with that form and period of the affection when the several muscular movements have been differentiated, and there is no longer a single and uniform response, but each department of the body liable to chorea responds after its own manner to a disorder which is now first mentally apprehended and intimately connected with emotional exaltation. As to the limbs there is no longer a mere restlessness impartially distributed, but complicated disorder of movement in which paresis and incoordination are variously blended, and which select by well-marked preference those muscles which are the readiest exponents of emotion. And as to the heart, in place of disturbed action and rhythm there is the proper emotional response with which everyone is familiar in his own person, an acceleration, namely, and often knocking impulse as well.

[1] Ziemssen's suggestion as to "the influence of restless muscular action upon aortic pressure and the working of the heart" (loc. cit. p. 437), fails to apply, I am convinced by repeated observation, in explanation either of the heart's irregularity or of its rate. It is indeed at once negatived by the admission that the heart disturbance of chorea is in no direct proportion to the violence of the muscular disorder.

Now at both the periods we are now considering, in childhood and in youth, both with cardiac irregularity and cardiac acceleration, we have, or we may have, apex murmur. It has been shown already that for such murmur no explanation is applicable except the functional one. And indeed, in the case of choreic patients, at that time of life when the so-called anæmic murmur is common, I know not how it is to be determined whether valve defect is due to chorea or to anæmia. Perhaps the same causes may operate in both cases, and the explanation of Dr. Nixon of faulty adjustment of valve and ventricle may serve for both. But, apart from these, there are the children to be considered. And, if we are still to apply the argument that the heart symptoms of chorea are in constant harmony with the rest, it is necessary to remember that there is another element in chorea besides those that have been alluded to. I mean *paresis.*

The real character of this choreic paresis has been somewhat obscured, I venture to think, by the prominence that has been given to those examples of it which have chanced to affect one side of the body only, and thus to give promise of material for illustrating the morbid anatomy of chorea. I would only remind the reader in this place as pertinent to the present inquiry, that a strictly localised muscular paresis very often mixes with choreic restlessness in children; that it is often very sudden both in its access and departure, giving to the affection an aspect of deformity which of itself it has not; that it may be recognised especially in a droop of the wrist or a falling of one shoulder, or feebleness of grasp of one hand; that it selects this part or that according to no known rule, and may in some instances show so prominently as to give the patient the appearance of paralysis rather than of chorea. Now all that can be said of choreic paresis as we see it in the limbs may be said as well of choreic murmur as we hear it at the heart. Both are characteristic of childish or at least youthful chorea, both are sudden in their development, variable in degree, and apt to come and go without any other corresponding change in the character of the disorder.

If then weakness and relaxation of the papillary muscles be

admitted as a possible cause of mitral regurgitation, and if a debility of this kind may arise temporarily from tobacco smoking or nervous exhaustion, much more should it arise in chorea where, apart from muscular fatigue, there is a special liability to paresis extending, as it would seem, to all the muscles which the disease is able to affect. And observe that this relaxation on the part of the papillary muscles, which is common both to anæmia and chorea, and productive of regurgitation in both, is not of the same origin in the two cases, nor always accompanied by precisely the same signs. In anæmia it gives rise to an equable murmur which only ceases with the gradual recovery of the patient; but in chorea it is (or it often is) a variable and inconstant murmur which hardly repeats itself precisely for two consecutive beats, and which appears and disappears without apparent cause, as does the similar paresis of the voluntary muscles, with which indeed, as I have repeatedly noticed, it may be strictly synchronous both in its time of arrival and duration. Moreover, with a murmur having this origin, we are rid of the difficulty which attaches to the doctrine of a fatigue paresis, and would require that the valvular defect should be in direct relation to the duration and severity of the chorea, which would associate it therefore, not so much with childhood, as with that violent form of chorea which is commonest at a later period of life.

Yet still the old objection will recur. Whence comes it that a temporary paresis of the valve apparatus is found to be associated after death, if not with endocarditis, at all events with a deposit of recent lymph on the edges of the mitral valve? That this appearance does not represent a true inflammation of the endocardium is rendered probable by its bead-like arrangement and limitation to the margin rather than the auricular surface of the valve. There is never ulceration, and where time has been allowed to elapse between the chorea and death, the valve is found to be in a normal condition. Only in cases where death supervenes either in the course of chorea or very shortly afterwards is this appearance met with, and in these not always. There is ample clinical and post-mortem evidence that it is neither lasting

nor injurious.[1] Mindful of this strict limitation of the facts to be interpreted, and of the necessity in the last resort of reaching a solution upon the principle of exclusion, the question suggests itself whether an imperfect closure of the mitral orifice, or rather such uncertain action of the valve segments as closes it at one time but not at another, might not, at the approach of death, and when with the flagging action of the heart fibrine is very readily deposited, so determine the seat and method of such deposit, as to produce at the edge of the valve the bead-like appearance that we see; the condition which immediately precedes death being one of the essential factors of the occurrence.[2]

It may be said, indeed, that whatever is true of the dynamic murmur of chorea must apply equally to the same murmur in anæmia and elsewhere. But of the post-mortem appearances of simple anæmia we know absolutely nothing. There is, however, a disease having a striking likeness to chorea in respect of its preference for the female sex, near connection with hysteria and with mental emotion, and apparently anomalous blending of functional with organic disturbance. In exophthalmic goitre we have at first acceleration of the heart and afterwards mitral murmur, a murmur which sometimes wholly disappears with the complete recovery of the patient, and sometimes is the precursor of organic mitral disease.

In conclusion, I would briefly recapitulate the facts which

[1] I venture to extract the following from a paper of my own, already referred to, on 'The Statistics of Fatal Chorea:'—

This post-mortem appearance occurs equally in those that die in connexion with chorea, and in those that die directly of it. Thus of Dr. Dickinson's 22, there are, as I reckon, 12 dying *with* but not *of* chorea; of these 10 have mitral valve vegetations and 2 have not. Of the 10 dying *of* chorea, 7 have vegetations and 3 have not. In Dr. Peacock's 3 fatal cases, all dying *of* chorea, 2 have vegetations and 1 has not. Out of 5 cases, the only examples I can find of fatal chorea with perfectly healthy heart, all save one, an old woman, are typical instances of death in and by chorea.

[2] The suggestion in the text is in fact that of Dr. Dickinson. "The beads," he writes, "are usually confined to the inner surface of the mitral valve and arranged along the attachment of the thin edge, where a line of minute but abrupt prominences is presented to retrograde blood, but an arrangement of more gradual slopes to blood flowing normally. Thus possibly the collection of fibrine is the consequence, not the cause, of the regurgitation." ('Pathology of Chorea,' loc. cit. p. 37.)

it has been the object of this paper to establish, together with the hypothesis which they seem to support. The facts are that in chorea the heart is apt to sympathise with the voluntary muscles at all ages up to the adult period, this sympathy being shown as well by dynamic murmur as by accelerated action, unevenness of rhythm, and, not seldom, the excited impulse common in hysteria, the particular manner of response being dependent upon the age of the patient. The hypothesis is that these several modes of heart affection correspond with as many modifications of chorea, which are exhibited not in the heart only, but in the voluntary muscles as well; these several regions sharing jointly, each in its own degree and after its own manner, in a disorder the area of whose influence is co-extensive with that of ordinary emotional disturbance: and, particularly that in all such variations the motor element of the affection is represented mainly by irregularity and unevenness of cardiac rhythm, the emotion element by acceleration, and the paresis element by dynamic murmur.

VISIBLE MUSCULAR CONDITIONS AS EXPRESSIVE OF STATES OF THE BRAIN AND NERVE CENTRES.

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(Continued from page 495, Part XII.)

SOME characteristic passive positions of the hand were described in the last paper as types; one other position must now be referred to—the "ortho-extended hand." Here the axis of the metacarpal bones and of the digits are in the same plane with the bones of the forearm. This is the position in which a strong man naturally holds out his hand when requested to put it forward. This may be considered as the antithesis of the "hand at rest." It was seen in Case V. *a*, presently to be described, in the left hand, when he held out his hands, while the right (paralysed) hand assumed the "nervous position."

A healthy child when it runs in its play, or to meet a friend, commonly holds out its arms, the hands assuming the "energetic position"; and this, as the result of observation, is I think characteristic of a healthy nerve-muscular condition; healthful children in pictures by the best masters are often thus represented. When a child is in the state of convulsion, it is well known that usually the hand is closed with the thumb turned in upon the palm, the fingers being flexed around it. Such a position of the hand in a child when ill is very indicative of a state of the nervous system predisposed to convulsions.

CASE X.—A child two years of age was admitted into Buxton Ward at the London Hospital, having had strong convulsions the day before. The child looked very ill, its

eyes were sunken, the body generally was rigid. The position of both hands was alike; the wrist was slightly extended, the bones of the thumb ortho-extended, or slightly more extended from the force with which its tip pressed against the last node of the index-finger. The metacarpo-phalangeal joints of the index and middle fingers slightly and equally flexed, the internodes remaining ortho-extended. The ring-finger rather more flexed than the middle one, separated from it, and the nodes slightly flexed. The little finger was in contact with the ring-finger, and assumed a similar position. I took a cast of the right hand, and the description at the same time. In a piece of sculpture exhibited at the Royal Academy two years ago a child's hands were represented so nearly in this character as to be very suggestive that the child was going off into fits.

The following cases may help to illustrate the advantage of studying common positions of the hand, that we may be able to detect others characteristic of weakness or disease.

Case V. *a.*—The notes of William D. have been already partially given; I now have to speak of the condition of his upper extremities. It has been said that he had been attacked with right hemiplegia; after a few months' treatment the condition of the arm improved, and the grasp of the hand became fairly good. In holding out both hands the left was straight with the forearm (ortho-extended), and the thumb straight with the fingers. In the right hand the wrist drooped a little, with slight extension of the metacarpo-phalangeal joints, and the thumb was extended—the nervous hand. There were some irregular and involuntary finger movements; these were chiefly flexor-extensor, occasionally slight abductor-adductor movements intervened, separating the digits. The muscular sense of this extremity was more interfered with than its muscular power.

Hemiplegia: involuntary movements of arm and face.

Case XI.—Arthur T., aged 10 years, came under my observation, May 1880, as the subject of a chronic left hemiplegic affection. His general health had been good, and the only

probable cause of brain disease was found in the fact that his father is syphilitic. His left upper extremity was wanting in muscular voluntary power and in muscular sense; certain involuntary movements of the hands and of the fingers were also observed. I now collect the notes describing the nerve-muscular condition of the hand. The left hand, when held out, assumed on the average the "nervous position"; not so the right, which was natural. A certain amount of involuntary movement of the fingers was seen; the middle finger moved the most; during the examination, and at other times when his fingers were curling up against his will, he would frequently use the right hand to straighten the fingers of the left. As to the kind of movement, it certainly was not voluntary, and on several occasions a similar movement was seen repeated. The dynamometer showed the power of the right hand as 47, against 36·5 on the left side.

I had often noticed that the boy looked much distressed when I examined the left arm, and this appeared not surprising considering that, with good bodily health, he was losing the use of the left arm, and at the same time there was family trouble from the father being out of work. Looking at the lad's face, one observed distinct over-muscular action in the upper zone, causing in the middle of the forehead transverse and vertical furrows, an appearance commonly resulting from the condition of brain associated with grief. For a long time I was deceived by this face, and thought the boy depressed, but later, on cross-questioning him, and inquiring of his mother, he appeared not only unconscious of these movements, but not to feel any mental distress. I was therefore compelled to regard the facial movements, which were suggestive of the expression of distress, as analogous to those of the fingers, which more resemble athetosis than any other phase of involuntary movement. The movements of the fingers were slow, involuntary, and unconscious; no child in chorea puts up his hand to straighten out the fingers which have curled up on the other hand. There were no general twitching movements, and those movements that did exist were not twitching in character. These points appear to indicate an analogy to athetosis, rather than to chorea, both in the hand and in the face.

CASE XII.—*Hemiplegia, malingering.*—Charles Giles, a country-looking man, aged 29 years, was an in-patient at the London Hospital, December 1880. He was admitted for right hemiplegia with anæsthesia. Suspecting the man a malingerer, I carefully noted the following points. "The right upper extremity is held out well and firmly, on a level with the shoulder; it is apparently easily kept out, and well sustained, as no muscular tremor or twitching is seen. The hand droops vertically from the wrist, the joint being flexed at about a right angle, and all the digits in a straight line with the metacarpal bones." This drooping of the wrist, as if hanging dead at the end of an active arm, is a condition totally different from the more or less flexed position usually seen in hemiplegia. He represented an arm without tone in the extensors of the hand and digits, and without rigidity. The hemianæsthesia was easily disproved; the electro-contractility of the two extremities was equal; he was sometimes caught using the right hand, and finally ran away, having inquired as to the locality of another hospital.

Before passing away from the consideration of the upper extremity as an index of brain conditions, we must notice the more active conditions of muscular movement. Equally characteristic with the passive positions of the hands are the muscular movements of the fingers,—twitching, tremors, and rhythmical movements. These conditions have been more fully studied and described than the characteristic passive positions (postures), and they have long been considered as visible muscular conditions expressive of the states of the nerve centres. The varieties of finger-twitching may be described as:—

(1.) *Flexor-extensor;* the primary movement being that of flexion, followed by a secondary extensor movement. This may be seen in a variety of cases, and in particular is seen in what is called "picking the bedclothes" in the typhoid state preceding fully developed coma.

(2.) *Extensor-flexor;* the primary movement being that of extension, followed by a secondary flexor movement. This is common in the slighter forms of chorea and in nervous

children; such twitches usually constitute the subsultus tendinum so indicative of exhaustion in the course of typhoid fever.

(3.) *Abductor-adductor* twitches; the movements consisting in lateral separations of the fingers, followed by their being drawn together again.

As to "finger-twitching," the "nervous hand," and "the relaxed orbicularis oculi," the following statistics are from an analysis of 34 cases from my note-books of the East London Children's Hospital:—

Finger-twitchings in 19.—Twitchings alone	in	8	cases.
With the nervous hand	„	8	„
With relaxed orbicularis	„	3	„
		19	„
The nervous hand in 19.—Nervous hand alone	„	7	„
With twitchings	„	8	„
With relaxed orbicularis	„	4	„
		19	„
Orbicularis relaxed in 10.—Orbicularis relaxed alone	„	3	„
With the nervous hand	„	4	„
With twitchings	„	3	„
		10	„

As to the general character of this group of 34 nervous cases in which nerve-muscular signs were specially noted, no cases of known organic brain disease were included, and all were under 15 years of age. I have abstracted and summarised the diagnosis of the 19 cases in which the "nervous hand" was seen:—"Headaches," 6; "Neurotic temperament," 3; "Anæmia and headaches," 2; "Headache and somnambulism," 1; "Restless sleep," 1; "Laryngismus," 1; "A dull child with congenital ptosis," 1; "Old rickets," 1; "Debility," 2; "Slight chorea," 1. The cases of "finger-twitching" had the same general characters as those with the "nervous hand," therefore I do not further describe them.

As to the kinds of finger-twitching, the varieties were noted as follows:—Simple twitching, 9; flexor, and abductor-adductor, 5; flexor, 3; abductor-adductor, 1; extensor, and

abductor-adductor, 1. As shown in the tables above, the "nervous hand" was associated with "finger-twitches" in 8 cases.

In cases where the right and left hands were compared, we find a difference in 6 cases, always to the disadvantage of the left hand; it specially presented "the nervous position" in 4 cases, and finger-twitches were specially marked on the left side in 2 cases.

The cases with "relaxed orbicularis" were specially marked by recurrent headaches, some with optical illusions and scarlet zigzag forms.

In 2 cases herpes zoster occurred while under observation.

The various positions, and movements of the head upon the spine, now claim our attention in as far as they depend upon and indicate the condition of the nervous system.

If we describe two axes of the head: (1) Interparietal, passing from one side to the other above the ears; (2) Occipito-frontal, passing from the occipital protuberance behind to the centre of the forehead in front, we can by referring to the positions of the axes define all the positions and movements of the head.

A. *Flexion and extension*—i.e. bending forwards and backwards of the head. Here the occipito-frontal axis has its anterior or posterior extremity depressed, but there is no deviation to either side, and the two ears remain at the same level.

B. *Rotation*—i.e. rotation of the occipito-frontal axis in the horizontal plane, the head remaining erect, and the interparietal axis horizontal, without flexion or extension.

C. *Inclination*—i.e. depression of one or other extremity of the interparietal axis, in which case one ear is lower than the other. Inclination may occur without either flexion or rotation, but is commonly associated with both. Right inclination means depression of the right extremity of the interparietal axis; left inclination, depression of the left extremity.

By means of these three terms we can, I believe, define all the possible positions and movements of the head. No account

is here given of the alterations of the positions of the head due to movements of the cervical spine. Flexion and extension of the head are of frequent necessity and physiological import; rotation is frequently necessary to move the eyes towards an object looked at, or to direct the ear towards a source of sound.

Inclination is, I think, often of other significance; it often indicates weakness, it is more often seen in young girls and weakly persons than in strong men. Inclination, with rotation to the same side, with slight flexion, is a position of the head very commonly seen in choreic girls, and then often accompanies an awkward ill-balanced position of the spine.

Inclination of the head, with rotation to the opposite side, and slight extension, is indicated by Sir Charles Bell in his figure in Adoration. In seeking general information as to the signs of cerebral adequacy in an infant, that is, whether the infant has a good sound brain, probably capable of due development, we always notice whether it can hold its head up, whether it usually keeps it erect when sitting up. In drawings of babies, we often see them represented as weak, evidently incapable of holding the head erect, the artist representing the want of strength of infancy by personal weakness and debility; this may be seen on comparing certain modern paintings with some of Raphael's infants.

An example of abnormal position of the head is the extreme retraction often met with in infants in brain disturbance, and also in cases of epidemic cerebro-spinal meningitis in adults. In all cases it depends upon tonic spasm of the extensors of the head, and is of grave import as indicating brain disease.

Case XIII.—January 1879. George W., age 4 months; complaint was made that he was stiff and unable to move his head. Fourteen days previously he had been taken ill suddenly with slight feverishness, and in a few days he could not sit up, and he fell into the condition in which he was when seen. He could move his arms and legs, but his head was distinctly retracted, and any movement of the head caused evident pain; there was no tonic spasm except in the muscles which retract the head; he looked intelligent, and the abdomen was natural. The tonic spasm lessened in a

week and passed away in three weeks; he remained two months under observation and recovered completely.

Such cases of retracted head are not uncommon, I think most of them die; it seems certain that in all cases the spasm depends upon nerve stimulus.

A curious and, as far as I know, unexplained chronic movement of the head in flexion and extension is occasionally seen in infants; the head constantly nodding up and down—a similar rotatory movement sometimes occurs.

Case XIV.—Elizabeth C., age 11 months, appeared healthy and well, and there was no complaint made except as to the head movements. These movements had commenced about a month previous to the time of observation; at first the mother did not notice them much, and there were never any signs of pain or general disturbance, the limbs were in no way affected. The infant looked healthy and well: the head-movements were simply rotatory in character; they came on, lasting a few seconds, then passed off again, leaving the head quiet, with the power of natural movement; then again three or four lateral oscillations succeeded, apparently never any vertical ones. There were never any general convulsions; no cause was assignable, and there was no history of injury or exposure to cold. The case came under my care at the Children's Hospital, Birmingham, in 1876.

Case XV.—Margaret McC., aged 8 months, seen April 1, 1879. She was brought under observation on account of constant nodding movements of the head. The movements were simply in the vertical direction, extension and flexion, being a series of nodding movements; these had continued for three months. The head was not retracted, the sutures appeared normal; the ribs were slightly bended. General health was good. The child continued under observation a month, the head-movements remaining unaffected; light appeared to increase the movements. These movements appeared somewhat analogous to those of nystagmus.

In cases of cerebral hæmorrhage, softening, tumour, and other coarse brain-disease, paralysis of one or more cranial nerves often forms a marked diagnostic symptom. As has already been said, facial paralysis from brain disease presents

certain characters distinguishing it from a lesion of the facial nerve. Ptosis and strabismus are not uncommon results of coarse brain disease; these facts teach us to look to the condition of the muscles supplied by the cranial nerves when examining the condition of the brain.

The first two cranial nerves are purely sensory, having no motor function; the third, fourth, and sixth supply the eye-muscles; of the movements of the eyes as indicating brain conditions much has been said already. The seventh nerve supplies the facial muscles, and its action has been discussed. There remain then for consideration the motor division of the fifth nerve (masticatory), the eighth, and the ninth.

Fifth nerve.—Tooth-grinding is produced by the action of the deeply-situated pterygoid muscles; champing of the jaws and trismus are produced by the masseter and temporal muscles; all these muscles are supplied by the fifth nerve, and it is to their condition that we must look for information as to the condition of the central origin of the nerve. Tooth-grinding when it has become a habit is indicated by the flattened condition of the tips or edges of the teeth which may be ground down—a sign that may be particularly seen in the incisors and canines. Ground teeth are very common in nervous children, such as those who suffer from recurrent headaches, restless sleep, somnambulism and finger-twitching. In lunatic asylums and wards for imbeciles it is very common to hear tooth-grinding on every side; in such cases tooth-grinding is a sign of central irritation of the fifth nerve; it is well to bear in mind that the sensory branches of this nerve supply the membranes of the brain and the external parts of the head.

Trismus is tonic contraction of the masseter and temporal muscles which close the jaws, and it is a common symptom in tetanus, epilepsy, and hysteria. Seeing that the slight disturbances occurring during sleep in many children caused the pterygoids to contract rhythmically, it is not surprising that grave disease should cause spasm of the other muscles supplied by the fifth nerve.

The ninth nerve is motor to the tongue, and this organ being a mass of muscular fibres running in various directions almost unsupported by bones, is very sensitive to changes in

the nerve-centres. In chorea the tongue is often jerked in and out in a manner quite characteristic of the disease; in other cases it is easily kept protruded, and its substance is seen to be in a condition of constant movement. Such irregular movement is very common in nervous children; a tremulous tongue is characteristic of alcoholism, and general paralysis of the insane. In hooping-cough we see tonic spasm of the tongue causing its violent protrusion, which is frequently so strong as to cut the frænum by stretching it over the lower incisors; the centre of the tongue is depressed, while its margins and tip are raised. Defective speech is not necessarily a lesion of the muscle of the tongue; but we can only know the condition of the centre of speech through its power to guide the muscles of the tongue and others used in speech.

Concerning the eighth pair of nerves, the distribution is partly to voluntary muscles, but the pneumogastric is distributed to many organs, and largely to organic muscular fibres; this fact brings us to the consideration that the tone or action of organic muscular tissue may indicate states of the nerve-centres. As the examples to be quoted are not cases of the visible action of muscles, they will only be enumerated. Palpitation may occur without disease of the heart, the action may be irregular, as the result of chorea or of coarse brain disease; the controlling action of the nerve over many glands and organs is probably due to its action upon the muscular walls of the vessels; there is also evidence that in asthma, bronchial spasm is due to irritation of the pneumogastric; in functional aphasia, the recurrent branches do not convey the due stimulus.

It is a matter of very common experience that children and adult patients "hold themselves awkwardly;" stoop, or otherwise give the spine and trunk an ill-balanced position, due to want of nerve-muscular energy, and characteristic of the condition of exhaustion and weakness. Doubtless there is much expression in a torso; in many cases a weakly condition is indicated by a stooping attitude, with a lolling over to one side. It is difficult to indicate in precise terms the positions and movements of the spine, and perhaps this is one reason why so little is known about the action of the muscles of the

back. In stooping, the spine may remain symmetrical, the bends of the column being only in the antero-posterior median plane, and not deviating laterally.

We may then distinguish symmetrical from asymmetrical positions and movements of the spine. Some further remarks will be made on this subject under the head of chorea.

As to the lower extremities, I have fewer observations to refer to than with the upper limbs. The gait and manner in walking may be characteristic of brain or cord disease. Conditions affecting the muscles of both lower extremities are usually dependent upon disease of the cord, and most of the signs by which spinal disease may be localised are derived from examination of the muscles of the legs. Cramp in the feet and legs is due to tonic spasm of the muscles, and probably is usually produced by overstraining of the muscles, or by indigestion, &c., i.e. it is not dependent directly upon the condition of the nerve-centres, and therefore is not to be taken as a direct index of their condition.

Reflex actions are used as means of localising and ascertaining the condition of nerve-centres. Here, again, visible muscular conditions are the indices; but on this subject no more will be said now, as it hardly comes within the scope of this paper.

As to the proximate cause of the passive positions or postures of extremities—the hand, the spine, the face, &c.—it is the relative tone of the muscles involved that brings this about; mainly the relative tone of the flexors and extensors; and this relative condition of tonicity results, it is believed, from the condition of activity of the nerve-centres corresponding. Hence it is probable that in observations of the involuntary postures, or passive positions of the limbs, hands and digits, we may find indices which we may record in cases indicating the relative tonicity of groups and sets of muscles, and the conditions of the corresponding nerve-centres.

No doubt the position of the limbs is simply the position of the bones, and this is simply the result of the relative activity of the muscles acting upon them; the action of muscles is the cause of the posture or movement, and the posture or movement indicate the action of the muscles, and so likewise the

action of the nerve-centres corresponding; therefore the postures may indicate the state of nerve-centres.

In describing postures it is probably worth while to attempt greater accuracy by indicating the angles of flexion and extension; adduction or abduction in the different joints. The condition of the hand in ortho-extension is that of a balance of the flexors and extensors; in the "hand at rest" the flexors have the best of it, and the amount of hypertonicity of the flexors may be indicated by giving the relative angles of flexion at the different joints, the less this angle the greater the hypertonicity of the flexors, i.e. the angle of flexion and the hypertonicity of the flexors are inversely proportional. In the ortho-extended hand there is no angle of flexion or extension.

The principle of antithesis is used by Darwin to explain certain forms of expression of the emotions, and I think that every physiologist must grant the muscular indications of emotion are due to the state of the nervous system, its structure and its activity. The dictum then involved in the assertion of this principle is that opposite conditions of muscular action indicate opposite states of the nerve-centres, and conversely, that opposite states of nerve-centres will cause opposite muscular conditions. The most natural opposite conditions of the nerve-centres are activity and inactivity, or activity and exhaustion. It is not only in the case of motor nerve-centres that the nerves become so exhausted that they cannot repeat their proper functions. The eye, after it has been fixed on one strong colour becomes incapable of perceiving that colour longer till it has been rested, but it may readily perceive the complementary colour; again, after having looked long at a bright light, a black spot only is seen when we look elsewhere. The following appear fair examples of antithetical postures.

1. The erect figure—the stooping figure.
2. The nervous hand—the energetic hand.
3. The hand at rest—the ortho-extended hand.
4. Orbicularis oculi relaxed—the orbicularis energised in laughter.

Chorea has in this essay been many times referred to,

because it is a condition of brain which we know only through the effect produced upon the muscles of the brain, and this condition affords abundant examples of various forms of nerve-muscular movement. It will then probably serve my purpose if I enumerate some of the principal muscles and groups of muscles that may be affected. In different cases very different groups of muscles may be affected, thus indicating the very different brain areas that may be choreic. It is suggested that in studying a case of chorea we should try and indicate the extent of brain affected by specially indicating the choreic area. The following points then should always be looked for—as present or absent in any case described—and the order of invasion of groups of muscles or their recovery should be observed:—

1. In examining a case to prove the fact of chorea it is very convenient first to look at one or both hands, held out free and disengaged. The kinds of movements of the hand and fingers have been dwelt upon and described.
2. The upper and lower extremities present the greatest mass of the choreic movements. It is important to note whether the finer or coarser movements be the most affected; the amount of involuntary movement, and the power of voluntary act that is left.
3. Hemiplegic varieties are common; the least mobile side may be much weakened, though not much moved.
4. The face. Varieties in this group of muscles have been discussed.
5. The soft palate may present marked movements of an irregular twitching kind, the levator-palati muscles working distinctly. I do not refer here to the dragging of the palate by the choreic twitchings of the tongue, but to the primary twitching of the palatine muscles. In some cases the levators are distinctly seen twitching upwards. This symptom is often absent in chorea, and when seen, I have observed that it has usually passed off early.
6. *Tongue* may be markedly jerked in and out. When

protruded, it may present much movement, but still be kept out a fairly long time.

7. Eyes. Upper eyelids often strongly retracted.
8. Head in the active stage is often moved much. During convalescence, and when the active movements have passed off, a lolling of the head to one side is common; i. e. inclination with rotation to the same side, combined with slight flexion.
9. Spinal muscles and trunk are often affected, but how to describe their condition I do not know. The child often balances itself very ill, throwing the scapular and upper dorsal region too far back, and thrusting the pelvis too forward, the spine still remaining symmetrical. It has sometimes seemed to me that the awkward appearance was due to want of adaptability of the proper compensations in the movements of different muscles.
10. The respiratory muscles. These may be affected much or little; the alæ nasi muscles may be affected also.
11. The vascular centres. The heart's action is sometimes irregular.
12. Varieties.—Hemiplegic.
    Paralytic.
    Local chorea; in Nerves IX., V., &c.

The significance of the action of muscles as indicating brain conditions has long been dwelt upon by writers. Camper, who wrote in 1821, has shown how the Laocoon presents evidence as to how deeply the ancients had investigated the influence of pain as expressed in the figure and the muscles. In this group, "not merely does the face, but the arms, legs—in short, all the muscles of the body indicate anguish." Further on he quotes from the words of Paulo Somazzo's work, 'Dell' Arte della Pittura,' published 1531, in which he describes the influence of the passions upon the muscles of the face, and still more minutely the different postures and contortions of the body. Camper there complains that authors have usually either confined themselves to appearances, or have "reasoned metaphysically concerning the operations of the mind, without

attending to the physical causes of the changes produced by these operations, but in my opinion (that is, Camper's) speculations concerning the manner of the soul's working, or concerning the seat of the soul, are of no use to the artist. These belong to metaphysicians, who, by the way, lose themselves in a labyrinth of terms, or words with no definite meaning, without having in the least explained the action of this immortal principle upon the corporeal and mortal frame." Camper then proceeds to give examples of the conditions of the muscles as indicating conditions of the mind, and then says, "the observation deducible from these effects is, that in every emotion of the mind particular nerves are affected; consequently every painter ought to make himself acquainted with the construction and connection of the nerves productive of these changes."

John Bulwer in 1649 published his work entitled 'Significative Muscles of the Affections of the Mind.' He expresses his opinion that every motion of the mind is indicated by a corresponding motion of the muscles. This is the same idea as Camper expressed later, and as is now well known to physiologists, that all postures and movements are the result of changes in nerve-centres. To study the conditions of the mind it is necessary now, as in former times, that the positions and movements of the body should be largely observed. Bulwer says, "When we assent, affirme, yield, grant, vote, confirme, confesse, admit, allow, or approve of a thing, &c., we are wont to nod or bend our head forward, the naturall reason of which motion in these, seems an approving, which is made by the Imagination, seeing, or hearing, somewhat done or said which accordeth very well, and this power remaineth in the Braine or forehead part of the head where in the cell and Seat of the Imaginations lieth. When any of these things give it contentment, suddenly it moveth the same, and after it all the muscles of the Body." In all cases, after describing the expression of a state of mind, Bulwer endeavours to explain which muscles take part in the act.

If anything can aid our studies of man, the matter becomes of interest to several classes of writers, to all those who study the body of man as indicating the activities of this brain or

mind, and as giving the knowledge of the means whereby the idea of his mental states and feelings may be expressed. If the matters discussed in this essay are of use in this direction, they concern not only the physician, but also the artist.

It is the work of the painter and sculptor to express by form the conceptions he may conceive of the condition of men and women in certain conditions of mind, states of strength and weakness; expressions of mental and physical pain, states of rest or repose, feminine coyness and defiance; the poet has to describe all these things in words. Clearly, also, it is a mistake for conditions of the limbs characteristic of disease to be used in art as mere expressions of feeling, unless the feeling be the result of disease.

There is then in the subject before us, "the visible conditions of the muscles as expressive of the states of the nervous system," a field for observation and description in which the artist and the physician may work together, observing and analysing with as much exactness as may be, the mode by which the varying conditions of the brain and mind are indicated to our eye, and may therefore be described by words, or by drawing or sculpture. We must study man in all aspects of the case, and when we see in the face, limbs, or body indications of his brain or mental condition, we should analyse and describe—first, the position of features and parts as we see them, then the muscles which produce these positions or movements, knowing that the muscular condition which has produced the movements or positions is the result of the state of the corresponding nerve-centres. It has been said that a man's face is the index of his mind, and this is true, for all the varying changes of expression in the face (except those of colour) are due to changes in the facial muscles, and these solely depend upon changes in nerve-cells.

The knowledge that we already possess of the nerve-centres is from observation of the condition of the muscles. In a given case, comparing the state of the muscles during life as they may be affected with paralysis or spasm with the brain lesion found after death, and by collecting and comparing many cases, it has been found that destructive or irritative lesions of certain parts of the brain cause paralysis or spasm of a certain

set of muscles corresponding. It is probable then that by carefully continuing these methods of examination—that is, by describing with accuracy all states which are indicated by conditions of the muscles—we may add still more to our knowledge of the functions of different parts of the brain, and gain a further insight into the pathology of that large group of nervous centres termed "functional." And to conduct such inquiries is a proper work for the physician. The objects of this paper have been to stimulate observations, and to direct and guide inquiries by definite lines and methods of investigation. Descriptions and definitions of some characteristic positions of parts and conditions of muscles have been given to aid in the clinical record of cases. Practical points are to some extent insisted on as specially characteristic means by which we may judge of the condition of the nervous system or of some centres forming it.

Irregular muscular movements, such as different forms of spasm, tremors, tic, &c., have often attracted much attention; but of these nerve-muscular conditions we have nothing more to say here. When dealing with a nervous case, however obscure and difficult to describe, if there be any such spasms or tremors, &c., local paralyses or weakness, movements or special passive positions, the case may be characterised by the description of these signs, and such indications of the state of nerve-centres are worthy of clinical study.

AN ELECTROTHERAPEUTICAL SUPERSTITION: "THE GALVANISATION OF THE SYMPATHETIC."

BY A. DE WATTEVILLE.

UNDER the title of "Galvanisation of the Sympathetic" has been, and is being, accumulated a large amount of that kind of literature which has done so much to cast discredit upon the subject of electrotherapeutics. Erroneous physics, imaginary physiology, fantastic pathology are there worked by processes unknown to ordinary logic into a sort of mystical creed. This heirloom of Remak's Benedikt[1] of Vienna claims, in accents of which the passionate enthusiasm is only equalled by the absurdity of the arguments he used to defend it. The mantle of the prophet has fallen upon his shoulders! Neftel[2] of New York (translating Benedikt without acknowledgment) pronounces the talisman to be one of the greatest therapeutic discoveries of the century. Speaking of "the rigid form of Infantile paralysis," the author of "How to use a Galvanic Battery" quaintly remarks, that "if these cases depend upon adhesions or exudations in the medulla, their absorption may possibly be promoted by localising a voltaic current in the superior cervical ganglion of the sympathetic. There seems no doubt that such an application causes a dilatation of the blood-vessels at the base of the brain, and is likely therefore to promote absorption."

Not only the cervical, but the abdominal sympathetic, the splanchnic nerve[3] and plexuses are each to be singled out by

[1] 'Elektrotherapie,' 1st ed., 1868. 2nd ed., 1874.

[2] 'Galvanotherapeutics,' 1878.

[3] The following observation is adduced as proof positive "that with the galvanic current we are able to exercise a direct influence on the splanchnic

the current in order to display its magic powers for cure. All the known, and many imaginary, diseases of the encephalon are to be relieved by acting on the superior cervical ganglion —and, let us not forget, "the *negative* pole on the ganglion, the positive on the nape of the neck is the really curative arrangement."

Galvanisation of the sympathetic is likewise a specific in a very respectable number of spinal complaints, rheumatic gout, optic neuritis, spasms in every part of the body, &c. And how could it well be otherwise? For, as Neftel profoundly observes, "by galvanisation of the cervical sympathetic we may produce hyperæmia or anæmia in the vasomotor centre"—a process, by the way, analogous to that of a dog running after his own tail—and further, "in the inhibitory centre of reflex action of the spinal cord, in the convulsive centre, and consequently prevent or call forth epileptic attacks and other cerebral disorders." So much for the rationale of the process: we must all deplore, however, the absence of any explanation as to the comparative therapeutical uselessness of hyperæmia and anæmia in these very centres, produced by nitrite of amyl and ergot, for instance; and simply believe that vasomotor effects obtained by the galvanic agency possess some occult powers *sui generis* hitherto beyond our comprehension.

However extravagant the pretensions put forward by the galvanisers of the sympathetic, and puerile their attempts at giving an objective basis to their beliefs, it may be granted that their method of electrisation is often useful. We must not allow ourselves to shut our eyes to clinical facts simply because presented to us in an absurd language.[1] Still less

nerves." Neftel applies a continuous current of fifteen cells from the back to the prostatic part of the urethra: "in less than half a minute the surface of the body assumed a deathlike pallor from the tetanic contraction of the small arteries, and this was followed instantly by syncope."

[1] Professor Erb's sober remarks stand in refreshing contrast with the tone we are accustomed to find adopted. In his experience good results have been obtained, in several spinal affections, from galvanisation with one pole over the vertebræ; the other in the subaural position. So far from calling to his aid any problematical sympathetic or vasomotor effects to explain such results, he merely hints "that in this way, possibly, a better permeation of the cord by the current is secured."

should the more general interests of electrotherapeutics suffer from such a cause. But it behoves every one whose name is connected with this branch of medicine to utter no uncertain sound on this subject; and at the risk of incurring the charge of aggressiveness, ruthlessly expose the pseudo-physiological inanities which have so long been a grievous stumbling-block in the way of common-sense and true progress.

The first point we must define is how much of the current we use actually passes through the cervical ganglion. To determine this with absolute accuracy is of course impossible; but a knowledge of the ordinary laws of electrical diffusion in conductors is sufficient to enable us to form some idea on this subject. Some writers have gone so far as to deny the possibility of reaching the sympathetic at all with our currents; a statement which need only be adverted to here as an illustration of the singular want of physical knowledge, even among men of high standing in the medical profession.[1] Erb, Burkhardt and others have, besides, put themselves at the unnecessary trouble of demonstrating the conductivity of the sympathetic by galvanometric experiments.

The factors regulating the density of a given current through a given point of the human body are several: first, the surface of the electrode; second, the distance of the electrode from the structure to be influenced; third, the relative conductivity of the several tissues; lastly, the position of the second electrode.

In the process known as the galvanisation of the sympathetic,[2] one of the poles is placed below the ear over the seat of the superior cervical ganglion. It should be of small size, so as to obtain the greatest possible density of current. We cannot, however, prevent an enormous diffusion of the current to take place immediately beneath the integuments. The ganglion is somewhat deeply placed, and surrounded by

[1] The same view has been held with reference to the bony envelope of the brain spinal cord. How living animal tissues, soaked with a saline solution, could be thought to be insulators is difficult to conceive.

[2] Fischer has proposed to call it "of the neck," a name that has the merit of involving no theory, but is rather vague. I should prefer a designation implying merely the position of the electrode: *subaural* galvanisation is perhaps as convenient as any other.

muscles and blood-vessels—all better conductors than itself, so that but a very trifling proportion of the electricity reaches it. To this we may add here, by anticipation, that weak currents only are safe in this region, owing to their ready diffusion through the base of the brain, and the very unpleasant symptoms thereby produced.

The only position of the second electrode by which any notable concentration of the current could be secured is inside the pharyngeal cavity, as proposed by Gerhardt. But to my knowledge all the other observers place that electrode at the nape of the neck or at the manubrium sterni. Now this is, as far as the superior ganglion is concerned, absolutely as if the application was strictly unipolar, and the "indifferent" electrode applied to some distant part of the body. In other words, the current as soon as it reaches the better-conducting tissues under the integuments diffuses in *all* directions; numberless paths are opened before it, through each of which it circulates in quantities inversely proportional to their resistance. Now this resistance in each case depends upon two factors: the diameter (or sectional surface) of the path, and the specific resistance of the tissues which compose it. If we assume the latter to be a constant mean, it is obvious that the imaginary direct path, the sectional surface of which is represented by the outline of the sympathetic nerve, is very small compared with the aggregate of the remaining paths through which the current circulates.

Among the authors who have treated the subject before us, two entirely different ideas are constantly found intermixed by a sort of mental jugglery which is apt to deceive the unthinking reader into acquiescence. In discussing the curative value of the galvanisation of the sympathetic, we must draw the same strict distinction between the immediate ends we have in view, as when we consider the curative influence of any other therapeutical agent. Do we intend to act directly upon a morbid condition of the nerve itself; or do we intend to stimulate the functions of the nerve, and so indirectly influence the condition of some other organ? In the first instance, it is obvious that the weak currents that reach the nerve may perfectly be entitled to the possession of certain

therapeutical properties—exactly as small doses of many medicines do doubtlessly possess. But if on the other hand we pretend to obtain our object by calling forth the physiological activities of the nerve, we must use quantities considerable enough to obtain clear proofs of such an action. Now in the vast majority of cases galvanisation of the sympathetic is claimed by its advocates to act in the latter way, that is indirectly by inducing changes in more or less distant organs, such as the brain, the eyes, and other parts. Disease of the sympathetic itself is comparatively rare and usually marked by distinctive symptoms; this part of the subject is, however, beyond our present scope. The questions before us are therefore: What are the effects of a physiological stimulation of the sympathetic? Do we obtain such effects in the medical application of electricity to this nerve in the living man? Our answers need not occupy many words. Of the two chief functions of the cervical sympathetic, which are to constrict the vessels of the head and dilate the pupil, we observe neither the one nor the other in practice. The insignificant and even contradictory results of numerous observations on the effects of subaural galvanisation upon the pupil cannot be appealed to; for it is notorious that the excitation of any sensory nerve, such as is necessarily involved in percutaneous galvanisation, is enough to produce some dilatation of the pupil. Further, if it could be shown that the effects observed were due to the electrisation of the nerves it would but give a measure of the amount of vasomotor change produced at the same time in the brain. Now the most accurate observations with the pupilloscope are necessary to discover the faint dilatation that is said to occur. What sane being could then attempt to balance a whole scheme of therapeutics upon such a minute basis?

But again, strange to say, it is usually assumed that the effect obtained on the circulation of the brain is one of dilatation, not of constriction, of the arterioles. The curious hallucinations under which many electrotherapeutists labour on this as on other points have been well illustrated by the quotation above given from a noted expert in these matters. It is true that, on the ground of certain experiments, it has been held that the galvanic current applied to the nerve acted

differently from the faradic, which always causes contraction of the cerebral vessels. Onimus and Legros[1] say that the ascending current contracts, whilst the descending dilates the arterioles. But it must be remembered first that these authors hold peculiar views about the "peristaltic" action of the muscular coat of the vessels. Second, that accurate investigations by Professors Vulpian,[2] Chauveau,[3] Fischer,[4] and others have failed to confirm these results. Every kind of stimulation of the exposed nerve yields the same result, constriction of the blood-vessels; and percutaneous electrisation, in Vulpian's hands, has had no effect whatever upon the arterial tension. Chauveau has shown that descending currents produce rather more contraction than ascending currents, the explanation of the difference being plainly the relative position of the negative pole in the two experiments. Let us note here that mistakes have occurred as to the names "ascending and descending current" with reference to the sympathetic: its course being upwards, the descending current is necessarily the one in which the negative pole is nearest to the head. Two things are certain if, as alleged, any notable arterial dilatation occurred during subaural galvanisation: the first, that we should expect to find some evidence of it in the integuments; the second, that this effect would readily be explained by some reflex action from the centripetal filaments of the vagus or cervical cutaneous nerves.

Much labour has been expended over the investigation of the retinal vessels during galvanisation of the sympathetic. Here, at least, it was thought decisive evidence of the vasomotor effects of the process would be obtained. Beard and Rockwell devote a considerable space to the description of

[1] 'Traité d'Electricité Médicale.'

[2] 'Le Système Vasomoteur,' vol. i.

[3] Original communication in Teissier's 'De la valeur thérapeutique des courants continus.' (Paris, 1878.)

[4] Fischer ('Centralblatt,' 1878, p. 124) after many experiments has come to the conclusion that in order to obtain higher vasomotor effects, faradisation or galvanic interruptions must be employed in the case of the sympathetic. Prsewosky, however, has found that polarisation of mixed nerves had an effect on the temperature of the limb. With the negative pole it was slightly raised, with the positive slightly lowered; but I am not aware that these results have been since verified.

their results, which were contradictory.[1] No wonder since the vasomotor nerves of the retina do not pass through the cervical ganglion, but are contained mainly in the trigeminus! In all fairness it must be said that this is a fatal objection to the argument derived from such negative results against the possibility of reaching electrically the cervical ganglion in man.[2]

Among the curious traditional proofs of the value of the process faithfully preserved in electrotherapeutical treatises is the relation of one or two cases where the *arm* of the patient used to become covered with perspiration every time he was galvanised.[3] Apart from the curious localisation of this phenomenon, it is in addition precisely the opposite of what physiology would make us expect. The cervical sympathetic does not supply the arm, and its irritation should produce a diminution of the sudoral secretion. This leads us to consider a fact which is coolly ignored by most writers, that is the position of the other pole, which is usually applied to the nape of the neck at the level of the origin of the brachial plexus. This is surely quite sufficient to explain a great deal of the effects attributed to the galvanisation "of the sympathetic"! Indeed a great therapeutical importance is laid upon "the production of an- or katelectrotonus of the upper part of the cord" by some of the very staunchest believers in the peculiar efficacy of the subaural position of one of the electrodes. Another circumstance to be noted *en passant* is the absence of any definite rules given in books as to the side on which the sympathetic is to be galvanised in unilateral brain disease. For instance, it would appear that most electrotherapeutists apply the current on the same side as the paralysis, that is, "act on the cerebral circulation" of the sound half of the brain. Surely this is hardly rational, and can be only explained by a reference to the historical development of the question. Remak held that the sympathetic had most extensive functions, among which to keep up the muscular tonus

[1] "Much seemed to depend on the temperament and condition of the individual; what would cause contraction in one would in the other cause dilatation." And these inanities are gravely reproduced by other authors, with commentaries!

[2] See Buzzard, 'Practitioner,' Feb. 1873.

[3] Moritz Mayer, 'Die Electricität,' &c. Cf. Eulenburg, loc. cit. pp. 30, 31.

of the whole body.[1] Electrisation of the cervical sympathetic on the side of the paralysis was supposed, therefore, to act directly upon the paralysed limb. Later, when the discovery of the vasomotor system began to carry away pathological theories to the absurd length still fresh to our minds,[2] the rationale of the process was changed to suit the times, but the practice remained.

Remak himself started the theory of "indirect catalysis" in explanation of the curative influence of subaural galvanisation in certain cases. He supposed in several forms of brain disease that the lesions consisted in effusions or adhesive exudations which the vascular dilatation produced helped to remove—a method of argument, like the "post hoc," not rare in medical literature, and which consists in inferring the morbid condition from the very therapeutical hypothesis which it is supposed to explain, or *vice versâ*. The tradition has been but too faithfully kept up by the disciples, as we have already had the opportunity to point out. It is evident that in subaural galvanisation a large number of structures, each as fruitful a field for electrotherapeutic imaginings as the sympathetic itself, receive their share of the electrical influence. I have already mentioned that the effect of the second pole on the cervical portion of the cord is usually kept out of sight. But besides this, the vagus, with its branches (among which the depressor nerve, irritation of which would suspend the vasomotor tonus[3] over a

[1] We are still told that it is "the knowledge that the cervical sympathetic has a powerful influence upon the nutrition of the whole nervous system, which induces us to apply a pole to the ganglion cervicale superius." Now as physiology is absolutely silent on the subject (atrophy of the brain subsequent to section of the sympathetic being merely secondary effect of protracted disturbances in the circulation) this *knowledge* is either an assumption which we are free to accept or not; or at the best a hypothesis framed to explain the supposed clinical results of subaural galvanisation. In the latter case the argument is a simple *petitio principii*—a reasoning in a circle.

[2] Professor Vulpian's Classical Lectures on the "Système Vasomoteur" (1875), contain an exhaustive discussion, physiological and pathological, of the whole subject.

[3] Cyon ('Principes d'Electrothérapie,' p. 205,) who, as the discoverer of the depressor nerve, displays a very natural partiality towards it, tends to explain the assumed vasomotor effects of subaural galvanisation solely to an action upon this nerve. But it is obvious that currents strong enough to stimulate a nerve comparatively so distant, would act powerfully on the sympathetic as well.

The great Russian physiologist, ever ready to pour his withering scorn upon

very great area), and the base of the brain itself (the very structures, i.e. which are often supposed to be influenced indirectly through the sympathetic), receive their full share of the current. The importance of this diffusion of the current through various important structures, though occasionally recognised in principle in electrotherapeutical writings, is usually entirely lost sight of when we come to the clinical observations, and the indications for treatment. The effects observed on the heart's action—a diminution in the number of beats—are much more probably due to a stimulation of the vagus than of the sympathetic, whilst the tendency to sleep and giddiness are not ascribable to any, more than problematical, "vasomotor changes" ("anæmia or hyperæmia," believers are not quite sure which), but simply to some unknown, perhaps molecular, change in the brain-cells and fibres themselves. The sympathetic, like the vasomotor system, has truly become a refuge for the destitute of hypothesis, as Clifford Allbutt has happily remarked.[1] When it is objected to one of the adepts of the process that it is difficult to conceive how the stimulation of the cervical sympathetic can possibly effect the curative marvels we are bid to accept as due to its agency, it is triumphantly pointed out to us that the remarkable and extensive vasomotor and trophic functions of the nerve are quite enough to explain all, even "the absorption of adhesions into the medulla"! If, on the other hand, we object that we have no proof in experiments on the living man of the occurrence of such physiological effects as are readily observed in the laboratory, it is declared that of course it is wrong to expect almost homœopathic doses of galvanism administered to the nerve to have the same obvious results as an energetic

the pseudo-physiological sciolism of "les Electrothérapeutistes," indulges here in some pathological elucubrations very much in the style of those he abuses so roundly: "Most of these atrophies [progressive muscular atrophy, saturnine paralysis, &c.], arise from nutritive disturbances called forth by a spasm of the vascular muscles. These disturbances cannot be better arrested than by getting rid of the vascular spasm: the irritation of the depressor nerve, in paralysing all the vascular nerves of the body, must abolish any vascular spasm depending upon the sympathetic nerve." Irritation of the latter "could but increase the nutritive disturbance in the atrophied muscles, since it would necessarily augment the contraction of the vessels." . . . *Ipse bonus forsan dormitat Homerus!*

[1] 'Brit. and For. Med. Chir. Review,' 1871.

faradisation. We are left therefore to conclude that, probably owing to some form of neurility special to the superior cervical ganglion, it will react in a mysterious manner to the weakest currents, if only these be assisted by a vigorous belief in their efficacy. But we cannot do better than to conclude with a quotation of the author just named: "Here is a neck, and inside the neck is a sympathetic nerve-system; if we galvanise the neck, the current will flow somewhere about the tissues, according to their various conducting powers, and other physical conditions; therefore the sympathetic may probably come in for some of it, and we all know that a galvanised sympathetic causes contraction of the blood-vessels. Now there are blood-vessels all over the body, and in the brain, as elsewhere; so perhaps they will be kind enough to contract among others, and perhaps contracting [or dilating reflexly] may do the brain some good, though how, it is not clear. Meanwhile, if we see no change in the pupil, ear, or cheek, or but slight changes only, we must have faith and hope that more sensible changes are going on within. Surely this is the very midsummer madness of reasoning!"

THE LOCALISATION OF ATROPHIC PARALYSES.

BY DAVID FERRIER, M.D., F.R.S.

Motor paralysis may result from lesions of different parts of the neuro-muscular apparatus, each more or less distinctly characterised by special clinical features and concomitants. It may be a consequence of destructive lesion, temporary or permanent, of the cerebral motor centres, or motor tracts, or of the spinal centres and their centrifugal paths—the motor nerves, or of disease of the muscles directly. The forms of paralysis have been classified variously, e.g. cerebral, spinal, peripheral, and myopathic; or central, paralysis of conduction, and myopathic (Erb). For practical clinical purposes it is most convenient to differentiate paralyses according to the exact seat of lesion, whether in the brain, spinal cord, or peripheral nerves, &c.; but in reference to the question immediately under consideration we may divide paralyses of nervous origin into two great divisions, cerebral and spinal. Under cerebral we may include paralysis from lesion, either of the motor centres themselves, or of their conducting paths, whether in the internal capsule, crus, pons, or spinal cord. Under spinal we should include only paralysis from lesion of the anterior horns, or their conducting paths, viz. the peripheral motor nerves.

This division is made, because, altogether apart from the clinical features of each case, in the one group the paralysed muscles retain their normal physiological properties, and emaciate only from disease; in the other the muscles undergo degeneration as well as wasting.

In an ordinary case of hemiplegia depending on disease of the cortex or internal capsule, or the pyramidal strands, the

muscles, though paralysed as regards volitional activity, still retain their contractility and normal reactions to galvanism or faradism, and waste only from long-continued inactivity. If artificially stimulated, they may be maintained in an active state of nutrition for an indefinite period, subject to certain secondary changes due to spinal complications.

On the other hand, in cases of lesion of the anterior horns of the spinal cord, as in poliomyelitis anterior, infantile or adult, or in cases of lesion of the motor nerves of the muscles, not only are the muscles paralysed, but they speedily lose their normal electrical reactions, and undergo atrophic degeneration.

The question is, why do the paralysed muscles undergo degenerative changes in the one case and not in the other? and further, why do certain muscles and muscular groups specially suffer from atrophic paralysis?

The trophic degeneration of muscles which ensues on destruction of the anterior cornua or division of the motor nerves, is considered by many as an argument in favour of the existence of distinct trophic nerves and nerve-centres whose function is to preside over and regulate the nutrition of the tissues in which they are distributed. The facts are unquestionable, but the explanation is purely hypothetical.

The researches of Waller, confirmed by all subsequent observers, show that the motor nerves degenerate when separated from the spinal cord; and the sensory nerves degenerate if separated from the ganglion of the posterior root, the degeneration taking place on the side of section, peripheral or spinal. Muscles also undergo degenerative changes when separated from their nerve-centres, or when these are destroyed; but not only do the muscles degenerate under such conditions, but also bones and glandular tissues. Trophic disorders also ensue on lesions of purely sensory nerves. Ophthalmia from certain lesions of the fifth nerve, is well known, and herpetic and other cutaneous disorders, glossy skin, &c., are familiar in connection with injuries of nerves. But though neither the coincident vaso-motor disturbances alone, nor the loss of protective adaptivity consequent on anæsthesia, nor the propagation of ascending or descending neuritic processes alone, may suffice to give a satisfactory explanation of the

phenomena, we are not necessarily obliged to postulate the existence of a distinct system of nerves and nerve-centres called trophic.

Sir James Paget, in his highly philosophical lecture on Elemental Pathology,[1] has ably urged the necessity of recognising "the distinction between the conditions of life and the essential properties of living things," and the advantages to be gained by the study of the diseases of plants, organisms free from the complications of a nervous system. "It is not to be doubted that an organism such as ours, in which the work is more divided according to its kind, and more distributed to appropriate organs, is more subjected to regulation by central organs, and the working of each part is more influenced by that of all the rest; yet it is not probable that in any instance the law is abrogated according to which each elemental structure lives its own life in a method determined by its own inherent properties."

As in plants so in animal tissues, the higher the organisation the less the power of independent vitality and adaptation, and the greater the tendency to degeneration when they are left to themselves and cease to form related units of a complex organisation.

Sigmund Mayer[2] regards the central nerve-substance, the peripheral fibre and its peripheral end-organs, as contributing "not only a functional but a nutritive unity," or a mode of viewing the facts which seems to me preferable to the hypothesis of special trophic nerves and centres. We have as units of external function certain nerve-centres, centrifugal nerves and peripheral organs, muscular or glandular, and their adjuncts. In union they exhibit certain vital properties and reactions which we call normal. The muscles react in a certain definite manner to stimulation, mechanical and electrical. But dissolve the unity, and the tissues are left to their own powers of nutrition. The muscles degenerate and exhibit different reactions to stimulation; there are disorders of secretion, and the bones and connective tissues alter their state of growth. The more specialised the tissue, the greater the

[1] 'Brit. Med. Journal,' Oct. 16 and 23, 1881.

[2] Hermann's 'Physiologie,' Bd. ii. part 1; "Specielle Nervenphysiologie."

degeneration, while the more lowly organised tissues proliferate and luxuriate. The nutrition and growth of the nerve-tissue itself is dependent on the ganglionic cell of which it is merely the prolongation, and the nerve rapidly dies when the connection is severed. Functional inactivity, the parts all retaining their normal connections with each other, leads merely to defective, not to altered nutrition. The difference is one of degree merely, not of kind. Here there is simple atrophy, whereas in the former case there is, as Mayer terms it, allotrophy.

The units of internal function are certain peripheral organs adapted for the reception of different external agencies, and the centripetal tracts which connect them with the nerve-centres in which they abut and excite action. The nutrition and growth of the sensory nerves and tracts, and with them the normal nutrition of the peripheral organs and their annexes, are dependent on the ganglionic cells situated either at the extreme periphery, as in the retina, or at a varying distance, as in the Gasserian ganglion of the fifth, and the intervertebral ganglia of the spinal nerves.

Sever the connection with the ganglion cells, or destroy them, and all the related parts (peripheral and central) undergo degeneration. The laws of nutrition of the sensory organs and their annexes are changed, and various disorders consequent on altered vital conditions tend to manifest themselves. Especially is this the case under conditions of irritation. The sensory nerves and tracts degenerate up to the point of their primary functional manifestation. Some of these run only a short course in the posterior root zones, others ascend a long course along the columns of Goll, and others again ascend along the direct cerebellar tracts of Flechsig.

A similar system of functional and nutritive units obtains in the central nervous system. The cortical motor centres and the pyramidal strands abutting in the anterior cornua of the spinal cord form such a system. Destruction of the cortex, or severance of the path at any point between the cortex and the multipolar cells of the anterior cornua, leads to degeneration from the point of lesion downwards to its termination.

The anatomical differentiation of the functional units of the central sensory system still requires investigation, though these

also will no doubt soon be accurately determined in the hemispheres and encephalic ganglia.

We may from this point of view clearly represent the essential difference between cerebral and spiral paralysis. The functional and nutritive unit in the one case is the grey matter of the cortex, pyramidal strands and their terminations in the anterior cornua. The process of degeneration when the continuity is interrupted stops at the anterior cornua, which have an activity altogether independent of cerebral influence, though occasionally they also become involved in the degenerative process. The functional spinal unit is independent of the cerebral, and hence the muscles maintain their nutrition according to normal laws, notwithstanding the withdrawal of the influence of the cerebrum. But if the multipolar cells are destroyed, or the motor nerve cut, muscular degeneration is a necessary result.

Atrophy with degeneration or allotrophic wasting of the muscles may in all cases be attributed to lesion in some portion of the functional spinal unit. It may be in the multipolar cells of the anterior cornua, in the motor nerves, or in the muscular tissue primarily. Of the forms depending on disease of the cornua we have poliomyelitis anterior, infantile and adult. Of the second form, the phenomena of peripheral paralysis, traumatic, rheumatic, &c. The central origin of bulbar paralysis seems well established. To which form, central, peripheral, or myopathic, we are to refer progressive muscular atrophy and lead-palsy, are questions on which some important differences of opinion exist. Though the clinical features and general pathological anatomy of anterior poliomyelitis are well known, yet the special localisation of the lesion in cases where not a whole limb, but certain muscular groups only are affected by atrophic paralysis, is a subject on which at present very little precise information exists. It not unfrequently happens both in infantile paralysis and atrophic-spinal paralysis of adults, that only certain muscles are affected, while all other muscles remain in a normal condition; and similarly in the lower extremity. Apart from actual demonstration of the seat of the disease, the absence of sensory impairment, the escape of other muscles deriving

their nervous supply from the same trunk, and the coincident affection of muscles supplied by several different nerve-trunks, all exclude peripheral paralysis, and point either to affection of the anterior cornua, or some of the motor roots of the nerves supplying the limb.

The question then arises, whether certain portions of the cervical and lumbar enlargements, or whether certain roots of the brachial and crural plexus have definite and specific relations to certain muscular groups, or whether there is perhaps a complete differentiation in both senses.

Of the various researches which have been made on the lower animals with a view to determine the distribution of the roots of the plexuses of the limbs, those of Peyer and Krause, on the brachial plexus of the rabbit, are most deserving of attention.[1] These investigators found that most of the muscles of the limbs were supplied by more than one root of the plexus. Krause in his work, 'Anatomie des Kaninchens,' gives a tabular view of the various muscles related to each root. It is seen, among other things, that the muscles nearer the shoulder are supplied by the higher roots, and those of the hand by the lower roots of the brachial plexus, the 8th cervical and 1st dorsal. The sensory roots have a corresponding superficial distribution over the regions of muscular distribution of the anterior roots.

These researches, taken in relation with others presently to be mentioned, have an important bearing on the question before us.

From a purely clinical and pathological standpoint, E. Remak[2] has recently advanced many able arguments in favour of the representation together in the grey matter of the spinal cord of those muscles which act in synergic combination.

Hence, for instance, the association of the supinator longus with the brachialis anticus and biceps, and the joint affection of these muscles along with the deltoid in what he calls his "upper-arm type" of atrophic spinal paralysis.

Besides the upper-arm type, he differentiates a "forearm

[1] For a more detailed account of previous researches on this question, see the communication by Dr. Yeo and myself in the 'Proceedings of the Royal Society,' No. 212; "On the Functional Relations of the Motor Roots of the Brachial and Lumbo-Sacral Plexuses."

[2] 'Archiv f. Psychiatrie,' 1876 and 1879.

type," which is essentially that seen in ordinary lead paralysis —paralysis of the extensors.

He has also attempted a differentiation of synergic muscular groups in the lower extremity; but the difficulties here are greater, both for want of material and in the way of exploration of the state of the individual muscles. But it appears that the sartorius frequently escapes, though the extensor cruris, supplied by the same nerve, is affected; and the tibialis anticus, according to him, not unfrequently goes with the extensor cruris.

With the object of throwing light on these topics by experimental research which might be considered as, with few exceptions, directly applicable to man, I have, in conjunction with my colleague Dr. Yeo, made a series of investigations as to the effects of irritation of the motor roots of the brachial and crural plexuses of the monkey. The results were embodied shortly in a paper read at the Royal Society.[1]

From these it is seen that each motor root represents a distinct functional combination; the muscles set in action being so correlated as to bring about a definite action clearly of an adapted nature. The relation is one of function, not of mere contiguity or community of peripheral nervous supply. Hence it is seen also that the purpose subserved by the plexiform junctions of the nerves of the limbs is to convey to the various muscles engaged in each functional synergy the requisite fibres related to each root.

The actions produced by the different roots may be briefly summarised as follows:—

Upper Extremity.

First dorsal;—action of the intrinsic muscles of the hand, muscles of ball of thumb, interossei, &c.

Eighth cervical;—closure of fist with pronation and ulnar flexion of wrist, retraction of the arm with extension of the forearm.

Seventh cervical;—the *scalptor ani* action, viz. adduction with rotation inwards and retraction of upper arm, extension of forearm and flexion of wrist and fingers so as to bring the tips against the flank.

[1] 'Proceedings of the Royal Society,' No. 212, p. 12.

Sixth cervical;—the movement of "attention!" viz. adduction and retraction of upper arm, extension of forearm, pronation and flexion of wrist, the palm of the hand being brought towards pubes.

Fifth cervical;—movement of the hand towards the mouth, viz. raising the upper arm inwards, flexion of the forearm with supination, and extension of the wrist and fingers.

Fourth cervical;—a similar movement of forearm and hand, but the upper arm is raised upwards and backwards.

The actions of the *lower extremity* are respectively:

Second sacral;—action of the intrinsic muscles of the foot, viz. adduction and flexion of the hallux, with flexion of the proximal phalanges and extension of the distal.

First sacral;—flexion of the leg, plantar flexion of the foot, flexion of all the toes at the proximal phalanges, and also of the distal phalanx of the hallux.

Fifth lumbar;—outward rotation of the thigh, flexion and inward rotation of the leg, plantar flexion of the foot and flexion of the distal phalanges.

Fourth lumbar;—extension of the thigh, extension of the leg and pointing of the great toe.

Third lumbar;—flexion of the thigh and extension of the leg.

The several muscles co-operating in these actions, so far as they could be observed and analysed, will be referred to subsequently.

Though the functional relations of the roots of the plexuses are indicated more or less precisely by our experiments, the question is not decided by them whether the motor nuclei of all the muscles engaged in each combination are in anatomical juxtaposition in the spinal segment corresponding to each root, in which case there would be several distinct nuclei for many of the muscles; or whether the motor nucleus of each muscle has a single definite position, not necessarily in the segment related to the root which calls it into action, but merely connected with the root by intracentral fibres running upwards or downwards as the case may be.

Thus the extensors of the wrist and-fingers are specially related to the fifth and fourth cervical roots, but they are also related to the eighth cervical, and the question is whether the nuclei are only in the upper segments, or also in the segment corresponding to the eighth cervical.

These questions scarcely seem to admit of solution experimentally, owing to the complications which would necessarily be encountered, but the clinical and pathological facts relating to anterior poliomyelitis are more likely to throw light on the point. In the absence of actual demonstration *post-mortem* we cannot be absolutely certain that in a given clinical case of limited infantile or adult atrophic paralysis, the lesion is in the anterior cornu and not in a motor root of the plexus. We must rely on the ascertained facts and principles of diagnosis of anterior poliomyelitis in such cases. These seem to me rather in favour of the view that the motor nuclei of the several muscles excited by each root are anatomically related to each other in the corresponding spinal segment, so that there may be several spinal nuclei of the same muscle.

If this is the correct view, it will follow that a lesion of the grey matter of the anterior cornu accurately limited to a distinct segment will produce the same effect as lesion of the corresponding root. Hence those muscles will be most affected by atrophic paralysis which are innervated entirely or almost entirely by this segment; whereas those which are represented also in other segments will be affected to a less extent. The degree of atrophy will be in proportion to the degree of innervation.

A pathological lesion will rarely restrict itself accurately to the segment corresponding to each root, and more commonly we shall have affection of more than one. But theoretically we may differentiate the types of spinal monoplegia into as many as there are spinal segments and motor roots.

From an analysis of the muscles involved in each of the combinations already referred to, so far at least as we have been able to ascertain them as yet, we may indicate the following as the muscles which are likely to be affected in poliomyelitis strictly limited to each segment; the muscles being placed in the order in which they will probably suffer.

First dorsal type ;—the intrinsic muscles of the hand, viz. muscles of the thenar and hypothenar eminences and interossei.

Eighth cervical type ;—long flexors, ulnar flexors of wrist, intrinsic muscles of hand, extensors of wrist and phalanges, long head of triceps (pectoralis major ?).

Seventh cervical type ;—teres major, latissimus dorsi, subscapularis, pectoralis major, flexors of wrist and fingers (median) triceps.

Sixth cervical type ;—latissimus dorsi, pectoralis major, serratus magnus, pronators, (flexor of wrist ?) triceps.

Fifth cervical type ;—deltoid (clavicular portion), biceps, brachialis anticus, serratus magnus, supinator longus, extensors of wrist and fingers.

Fourth cervical type ;—deltoid, rhomboid, supra- and infraspinatus (teres minor), biceps, brachialis anticus, supinator longus, extensors of wrist and fingers, diaphragm.

In the lower extremity we should have the following types :—

Second sacral type ;—intrinsic muscles of the foot, strictly parallel to the first dorsal type.

First sacral type ;—muscles of calf (plantar flexors), hamstrings, long flexor of big toe, intrinsic muscles of the foot.

Fifth lumbar type ;—flexors and extensors of toes, tibial muscles, sural muscles, peroneal muscles, outward rotators of thigh, hamstrings.

Fourth lumbar type ;—extensors of thigh, extensor cruris, peroneus longus, adductors.

Third lumbar type ;—ilio-psoas, sartorius, adductors, extensor cruris.

No absolute accuracy is claimed for this order of enumeration, but as a general indication it may be accepted provisionally as a guide to further more minute clinical research. And it will be seen by the cases given below that clinical observation confirms in a substantial manner the distribution of the muscular affection in some at least of the types indicated.

It is quite possible for slight affection of some of the muscles forming a group, and also innervated by other roots, to escape detection by electrical exploration, and it will be necessary in future inquiries to test more minutely the relative strength of

the individual muscles, and compare them carefully with those of the other side.

As has been indicated, we can scarcely expect to meet with pure types; more commonly we are likely to have a blending of two or more contiguous ones.

The following cases which have recently come under my own observation are examples of an affection more common perhaps than any other spinal monoplegia of the upper extremity, viz. of the groups innervated by the upper cervical roots.

CASE I.—Mary T., ætat. 45, a married woman, following the occupation of a sewing-machinist in connection with a tailoring establishment. This occupation involves holding and lifting heavy materials with the right arm.

A week before coming to the hospital—on November 13, 1880—she had had some pain in the right shoulder, but the movements of the arm were not affected. On Sunday November 12, on getting out of bed she found she could not raise her right arm.

On examination it was found that she was unable to abduct or raise the right arm away from the trunk beyond an angle of about 25°. Flexion of the forearm was very feeble. The arm could be flexed to a certain extent in the position midway between pronation and supination, by the aid of the muscles of the forearm attached to the condyles of the humerus. The shoulder could be elevated, but retraction upwards and backwards was very feeble on the right. Supination was much feebler on the right than left. The fingers could be clenched forcibly, and the wrist and fingers could also be extended. No comparison of the relative strength of the extensors was, however, made in this case.

The sensibility was in every respect normal.

The electrical reactions were tested on November 13, again on December 6, and on December 15. At the last date the condition was as follows:—

Circumference of upper arm.—Right, $9\frac{1}{8}$ inches; left, $9\frac{1}{2}$ inches.

Circumference of forearm an inch below condyles.—Right, $8\frac{3}{4}$ inches; left, 9 inches.

Acromio-axillary circumference.—Right, $14\frac{1}{2}$ inches; left,

15 inches, indicating wasting of the deltoid, flexors of forearm, and supinator longus.

The supra- and infra-spinous fossæ were also more distinct on the right side, and the muscles soft and flabby. Outward rotation of the right humerus impossible. Retraction and elevation of right scapula weak.

Faradic reactions.—Deltoid: excitability abolished in the scapular and acromial divisions, normal in clavicular portion. Excitability considerably diminished in the right biceps, brachialis anticus, supinator longus, rhomboid, and infra-spinatus: uncertainty as to the supra-spinatus, owing to the action of the trapezius.

No perceptible difference in the reaction of the serratus magnus of the two sides, nor of the extensors of the fingers.

Galvanic reactions.—Reaction of degeneration in the right deltoid (outer two-thirds), rhomboid, infra-spinatus and teres minor A C C > C C C. Excitability diminished in the biceps, brachialis anticus and supinator longus, but C C C > A C C.

Remarks.—This case diagnosed as one of limited anterior poliomyelitis corresponds almost in every detail to the fourth cervical type. The deltoid, the outward rotators of the humerus, the rhomboids, flexors of the forearm with supinator longus were distinctly affected, and in the proportion which one might expect, considering the degree of innervation of the respective muscles by the fourth cervical root. The extensors of the wrist and fingers were not perceptibly affected as regards electrical reactions, but their innervation by other roots, the fifth and eighth, would readily explain this.

Case II.—Daniel C., ætat. 65, a labourer, admitted at King's College Hospital in my out-patient department, on March 24, 1881.

The patient stated that a month ago on getting up one morning he found he could not raise his right arm. He had no pain in it. He had previously always enjoyed good health, and previous to his attack was not complaining specially.

The sensibility of the right arm was everywhere normal.

Abduction of the right arm from the trunk almost impossible. Cannot put his hand in the side pocket of his coat. Very feeble power of flexion of the right forearm. Extension

of the wrist and fingers of the right hand much feebler than on the left.

The electrical reactions on April 7 were as follows:—

Faradic.—Very feeble, almost abolished, reaction of right deltoid,—the clavicular and acromial portions being affected as much, if not more, than the scapular portion.

Excitability diminished in right rhomboid, infra-spinatus, serratus magnus, biceps, brachialis anticus, and supinator longus. Slight diminution also in the extensors of fingers and wrist.

Galvanic.—Excitability increased, but sluggish reaction, in the right deltoid, A C C > C C C.

Diminished in the right biceps, brachialis anticus, supinator longus, and extensors of the fingers, but C C C > A C C.

Remarks.—This case indicates an affection of muscles innervated both by the fourth and fifth cervical roots. The clavicular portion of the deltoid was here distinctly affected, as much if not more than the scapular. This portion seems specially in relation with the fifth root. The extensors of the fingers and wrist were also impaired.

The affection of the serratus magnus is also in agreement with what we should expect from the experimental data.

Case III.—Robert W., ætat. 17. Had worked as a plumber for a year. Came under my care at King's College Hospital on April 12, 1881. Nine months ago he began to have pain in his left shoulder and some difficulty in using his arm. This increased gradually up to the present condition.

Examined at this date, and again more thoroughly on April 28, and on June 13 the following condition presented itself:—

Considerable wasting of the left shoulder, upper and forearm as compared with the right.

Acromio-axillary circumference.—Left, $13\frac{1}{2}$ inches; right, $13\frac{7}{8}$ inches.

Middle of upper arm.—Left, $7\frac{1}{4}$ inches; right, 8 inches.

Forearm, 2 inches below olecranon.—Left, $7\frac{5}{8}$ inches; right, $8\frac{3}{4}$ inches.

Complains of pain in the left shoulder, but the sensibility is normal. Flexion of left forearm very feeble, L. = 12 lbs., R. = 50 lbs. Diminished power of extension of left wrist also

as compared with right, L. = 12 lbs., R. = 18 lbs. Cannot rotate left arm outwards. The inferior angle of the left scapula is more prominent and stands 1½ inch higher than that of the right.

Electrical reactions: Faradic.—Deltoid, much diminished generally. The clavicular portion reacts more readily than the rest.

Diminished also in infra-spinatus, serratus magnus, biceps, brachialis anticus, supinator longus, rhomboid, pectoralis major and latissimus dorsi.

The excitability was increased in the extensors of wrist and fingers.

Galvanic.—Deltoid: greatly diminished in all parts. The clavicular portion reacted more readily. No qualitative change.

Diminished reaction also in the infra-spinatus, biceps, supinator longus and the other muscles above mentioned. No qualitative change. Excitability of the extensors of wrist and fingers increased, but C C C > A C C.

Remarks.—This case is also a combination more or less complete of the muscular groups innervated by the fourth and fifth cervical, and also by the sixth. Nine months having elapsed since the onset, it is difficult to determine what the conditions of the respective muscles were in the early stages. But there were abnormalities, as above indicated, present in those muscles which we have seen act together on stimulation of the fourth and fifth and sixth cervical roots. In this case there was some suspicion of lead-poisoning. The patient had not, however, worked long as a plumber, he had never had lead colic, yet there was a suspicious-looking line at the margin of the gums. He had not had drop-wrist. The fact, however, is important, and will be referred to subsequently in connection with saturnine paralysis.

Case IV.—Sydney W., ætat. 15. Admitted as out-patient at the National Hospital for Paralysed and Epileptic on April 8, 1881.

On January 1880 complained for several days of pain in the left shoulder. In the summer the arm became weak, and has been more or less paralysed ever since.

Cannot raise the left arm from trunk, nor draw the left shoulder upwards and backwards. Flexion of left forearm very feeble. Cannot place his left hand on right shoulder; cannot cross his wrists behind his back.

There is wasting of left shoulder, hollowing of the supra- and infra-spinous fossæ, thinning and flabbiness of biceps and supinator longus. The lower angle of left scapula is more prominent than the right when the arms are hanging down by the sides. This becomes much more pronounced when the arm is raised.

The sensibility was normal.

Faradic reactions.—Deltoid, sub-normal in clavicular position, almost abolished in acromial and clavicular portions. No action obtainable in left infra-spinatus, very doubtful action in left rhomboid. The right contracted vigorously to same current. Diminished action in left biceps, supinator longus, serratus magnus, pect. major, latissimus dorsi. Slight diminution in the extensors of fingers.

Remarks.—This case is almost an exact repetition of Case III. —the upper cervical type.

These cases show that in addition to the usually recognised combination of affection of the deltoid, flexors of forearm and supinator longus, constituting Remak's upper arm type, other muscles also suffer more or less in accordance with the grouping indicated by experimental research. The outward rotators of the humerus and rhomboids go with the deltoid, more particularly when its outer two-thirds are affected, and the serratus magnus, which is a synergic muscle with the deltoid, likewise suffers, especially when there are indications of affection also of the clavicular portion of the deltoid. The escape of the clavicular portion of the deltoid, which is so commonly noted even when the scapular and acromial portions are affected, receives its explanation in the fact that it goes more with the fifth than with the fourth cervical root.

The extensors of the wrist and basal phalanges are also shown to be liable to be affected in combination with the supinator longus, flexors of the forearm and deltoid, though to a less extent relatively than these, and more particularly when the affection implicates both the fourth and fifth more or less.

The comparatively slight affection of the extensors even in this case seems to be satisfactorily accounted for by the representation of the extensors again in the eighth cervical root, as synergic with the flexors of the fingers. This fact would, if this is the correct view, support the notion that the motor nuclei of the extensors are not anatomically restricted to a single definite position in the anterior cornua of the cervical enlargement, but are represented in each segment corresponding to the motor root which calls them into action synergically with other muscular groups.

Otherwise it would be difficult to account for the relatively slight affection of the extensors of the fingers, when the other muscles of the groups are affected to a very marked extent, either on the theory of direct affection of the fourth and fifth motor roots, or on the more probable myelitic nature of the lesion in the above-recorded cases.

The above localisation affords an explanation also of the peculiar "main à griffe," described by Charcot and Joffroy in connection with hypertrophic cervical pachymeningitis. Here we have a very marked extension of the wrist and basal phalanges with paralysis and atrophy of the muscles supplied by the ulnar and radial nerves. This will occur when the pachy-meningeal lesion is limited more especially to the lower portion of the cervical enlargement, leaving the upper segments specially related to the extensors free, and hence the unantagonised action of these muscles and the characteristic "griffe," with hyperextension of the wrist.[1]

On the other hand, when the pachymeningitis affects the upper half of the cervical enlargement, as in a case reported by Dr. Leech, and figured by Dr. Ross,[2] we have exactly the opposite condition, viz. unantagonised action of the flexors and pronators, the muscles supplied by the musculo-spiral being specially paralysed.

[1] See figure in Charcot's 'Leçons.' Troisième Partie, 1874, p. 251.
[2] 'Diseases of the Nervous System,' vol. ii. p. 388.

(To be continued.)

Critical Digests and Notices of Books.

Dr. Leonardo Bianchi *on the Significance of Electric Excitation of the Cortical Motor Zone.*

From thirty-seven experiments the author comes to the following conclusions:—

1. That there exist in the cerebral cortex cellular excito-motor elements capable of being experimentally excited by electricity.

2. That these elements, if indeed they are to be found disseminated through the whole cortical grey substance, are yet grouped in such a manner in the cortical motor zone as to constitute of themselves alone true cortical centres, and the only ones which experiment has succeeded in discovering.

3. These centres are true psychic motor centres, distinct from the centres for reflex cerebral movements, and also from those for automatic movements.

4. It is in no way proved that the functions performed by the voluntary motor centres can be carried on by any other cerebral organs. On the contrary, this seems highly improbable.

5. It is equally a question whether these centres are composed entirely of motor elements, which receive impulses from other elements in which volitional determination and the idea of movement are produced, or whether these powers reside in them conjointly with the faculty of developing excito-motor force.

6. However this may be, the bundle of fibres proceeding from them pass exclusively into the internal capsule (its two anterior thirds); and a lesion of the latter produces a hemiplegia probably quite identical even in animals with that produced by destruction of the motor cortical zone.

7. It appears to be proved that the cerebellum is not directly concerned in the course followed by the electric stimulus applied to the motor zone, in order to produce the phenomena of which we have been treating.

The author, having already in 1878 reviewed the history of the experimental demonstration of the cortical motor centres, does not re-discuss this part of the subject. Neither does he consider the question of the production of movements through centres for organs of sense, confining himself to movements of the head, face, tongue, mouth, and limbs. The corresponding centres are located about the fissure of Rolando in man and apes; about the cruciate sulcus in dogs, cats, and sheep (though the sulcus is less marked in the last) in corresponding parts, although apparently in different localities in the solipeds; and in parts approximately correspondent in the rabbit, whose brain shows only a trace of the cruciate sulcus.

The first experiments were made with the view of testing the opinion that movements obtained on stimulating the cortical motor zone were due to irritation of the ganglia at the base of the brain. If this were so, the more nearly the stimulus approached these ganglia the more marked should be the movement. In experiments 1, 2, 3, the brains of two dogs and a rabbit were pierced by electrodes, convolution by convolution, and point by point, on the lateral parts of the hemisphere, and also (in the dogs) upon the internal aspect of the same, on the point nearest to the corpus striatum, but without the slightest effect being produced in any of the experiments. It was not possible to cause isolated movements of one limb or of distinct groups of muscles by irritating the corpus striatum directly.

Experiments 4, 5, 6, 7. On penetrating with fine platinum needles, covered as far as the point with a layer of vulcanite, to the caudate nucleus and, in a different direction, to the corpus striatum, there followed, not the definite movements of special parts obtained on stimulating the motor zone, but this: a sudden tonic contraction of all the muscles of the opposite side. The thigh flexed on abdomen with the limb and foot extended, the anterior limb carried forward, rigid; the trunk laterally curved as in pleurosthotonus, the head

turned to the opposite side. These movements differed in character from those got by stimulating the cortical motor zone, being more rapid or instantaneous, and not being apparently adapted to any determined end.

Experiments 8, 9, 10 and 11 show that the course taken by a stimulus commencing in the cortical motor centres is through the internal capsule. When this is divided, no movements follow, even although the basal ganglia and all their connections with the cerebral cortex remain intact. In one of the experiments, section of the capsule was not quite complete, and then movements in the anterior extremity followed stimulation of the cortical zone. These consequences of division of the internal capsule agree with the anatomy of the part, and in particular with the existence of the large pyramidal cells found in it by Betz and Mierzejewski.

Experiments 12 and 13 show that stimulation just in front of and just behind the motor zone is not followed by movements under moderate stimulation, although stimulation by a strong current may induce epileptiform convulsions.

Experiment 14 is very important. Dr. Bianchi chloroformed a dog, laid bare the motor zone, and obtained the usual movements. The animal meanwhile came out of the chloroform. On giving it more chloroform, the respiration suddenly ceased. He reapplied the current more strongly than at first, and obtained once only the same circumscribed movements. In the meantime, on attempting to revive the animal he found it dead.

The author says that this experiment proves that the animal could not have have had any sensation by which it retracted its limbs. It is known, he says, that in the deepest anæsthesia from chloroform or ether the electric contractility is little changed, and endures for some time even after death. All the nervous mechanism of sensation is paralysed long before the apparatus of motion, "and my observations," he says, "whose significance I cannot overlook, although they are opposed to those of Schiff, confirm definitely the fact that the electric irritability of the motor nervous apparatus resists narcotic influences much longer than the sensory apparatus."

In reference to this important statement, it may be observed that in giving patients chloroform for surgical operations or in

obstetrics, they continue to move, to flinch from the knife or the hand, and also to talk, laugh, sing or pray, long after they cease to know they are doing these things. At least, when questioned after their return to consciousness, they say they remember nothing of them. These facts seem to corroborate Dr. Bianchi's views. But, on the other hand, it should not be overlooked that another explanation is possible; this namely. It may be the case, as Dr. Munk suggests, that the *perceiving* centres are not the *remembering* centres, and therefore persons who do not remember saying or doing certain things may yet have perceived them when they did them. Dr. Bianchi's dog, in short, may have *perceived* or *had the sensation of* the electric stimulus by its perceptive centres, even after the *remembering* or *judging* centres were dead.

In experiment 18 he removed the grey substance of the cortical motor zone, and then by stimulation he obtained the usual movements, only more rapid and rather tending to be tetaniform. From this he infers that no sensation of contact can have preceded the movement of the limb, since the apparatus of sensation was removed. This is of course the case, but still it scarcely follows that there is no sensation when the apparatus is perfect, as in ordinary circumstances. All that he is warranted in inferring seems to be that the movements can be produced by stimulating lower centres than those of the cerebral cortex, a fact which, of course, is not new. But then the movements tend to become tetaniform. That is, on the opponent's supposition, just because the controlling sensation of contact is absent.

Dr. Bianchi says that tactile sensibility is not diminished by removal of the cortical zone. This may be, and yet it may be true that perception of that tactility may be not only diminished, but destroyed by that removal.

In experiment 19 the author destroyed the cortical motor zone in a rabbit, after having determined by the electric current the centres for the movements of retraction of the angle of the mouth. After removal it was found that the movements were no longer obtained. After that, he passed the pulps of the fingers over the ends of the hairs fixed around the mouth, and the rabbit frequently turned his head from the

opposite side, so affording the most indisputable evidence that the tactile sensibility was intact.

This experiment, the author says, is totally subversive of the reflex theory; but, supposing the *whole* centre to have been removed, would not passing the fingers *around* the mouth (intorno al muso) excite other reflex functions, besides the functions of those removed? And lastly, even if centres cannot act when removed, does it follow that they do not act in normal circumstances when the mechanism is perfect?

Experiments 20, 21, and 22 relate to the action of strychnine and lead to a discussion of the four propositions laid down by Brown-Sequard. 1. The symptoms in organic affections of the brain do not arise through the loss of a function belonging exclusively to the part destroyed, nor through the direct effect of a manifestation of the special property of that part. 2. On the contrary, these symptoms are only the consequence of an influence exercised upon other parts at a distance greater or less from the seat of the organic lesion visible at the autopsy, an influence caused by an irritation transmitted either by the very part destroyed or by the neighbouring parts. 3. Paralyses, amauroses, anæsthesias, aphasia, are produced by a mechanism identical with that by which the heart is arrested by irritation of the vagus. 4. The mechanism of the manifestations of exaggerated activity, delirium, epileptic and choreic convulsions, vomiting, &c., is the same as that by which all these phenomena are produced, since their original cause is a peripheral irritation to the skin, to the mucous surfaces, or to some point in the course of a sensitive nerve.

The author does not care to follow Brown-Sequard into the wide domain he occupies, but confines himself simply to the consideration whether there exists a motor zone in the cerebral cortex. This being so, the only question for consideration is whether the symptoms consequent on irritation or on lesion of the cortical motor zone depend on an influence exercised upon other parts at a distance from the seat of the irritation or visible lesion. The action at a distance may be conveyed either to other parts of the cerebral cortex, or to the ganglia and other parts at the base. But as in men and the higher animals the basal ganglia govern only automatic movements,

the action by other parts of the cortex is the only one to be considered.

In order to determine this question, experiments 23, 24, 25 and 26 were made. In these experiments a given centre, say that for the anterior limbs, was isolated from the rest by a platinum plate which could be heated by a galvano-caustic apparatus. This being plunged into the brain as far as the white substance and then heated to redness, effectually separated the centre from the others. But on reapplying the current, the accustomed movements occurred.

In experiments 27, 28, and 29 the same separation was effected by the knife, the animal being under the influence of chloral, but still the ordinary movements occurred.

Experiments 36 and 37 relate to the action of the cerebellum, and seem to the author to justify the conclusion already detailed as No. 7.

The remarks on Dr. Brown-Sequard's views are exceedingly interesting, but space forbids our entering on them. If the author has not solved the difficult questions connected with the discussion of cerebral functions, his experiments are well conceived and carefully executed, and may, on the whole, be said to have advanced our knowledge. A. RABAGLIATI.

BETZ *on the Structure of the Cortex Cerebri.*

IN an article contributed to the 'Centralblatt' for August 1874,[1] Professor Betz has given the details of an extensive examination of the cerebral cortex, which proved a valuable addition to our knowledge of its histology, as he therein advanced several original observations indicating the existence of certain definite regions hitherto unnoticed amongst the list of those characterised by a dissimilar lamination. With a few exceptions and misstatements, this article was a really fresh addition to existing knowledge of cerebral topography. In the March number of the same journal for the present year[2]

[1] "Gehirncentra." 'Centralblatt f. d. Medicin. Wissensch.' Nos. 37 and 38, 1874.

[2] "Feinere Structur der Gehirnrinde des Menschen." 'Centralblatt,' Nos. 11, 12, 13. 1881.

there appears an account of a still more extended inquiry into the diversified structure of the human cortex cerebri, which contains so much in which Professor Betz has been wholly anticipated by the labours of other inquirers without any recognition of such work, that we naturally wonder at the author's neglect in this respect. It is therefore thought advisable, in a critical survey of his two articles, to review the facts which have been advanced by others, and practically ignored by Professor Betz. In his earlier memoir the following conclusions are arrived at.

1. The whole cerebral cortex is divisible into a motor and sensory portion approximately divided by the fissure of Rolando.

2. The motor division is characterised by the prevalence of large pyramidal cells; the sensory by the prevalence of so-called granule cells.

3. A constant lobule exists in man and the higher apes upon the median aspect of the hemisphere at the extremity of the central gyri, which he names the paracentral lobule.

4. This lobule is characterised by the presence in its cortex of the largest nerve-cells of the brain, hence termed "giant pyramids."

5. These cells closely resemble the motor cells of the anterior cornua of the spinal cord.

6. Such cells are found in a similar position in man, chimpanzee, ape, and baboon.

7. They likewise exist in the gyrus surrounding the crucial sulcus in the dog. (Sigmoid gyrus.)

8. The size of this lobule bears a peculiar vicarious relationship to the development of the central gyri; the one being large and complex when the other is but poorly developed, and *vice versâ*.

9. This sigmoid gyrus (found electrically excitable by Fritsch and Hitzig) is not the analogue of the central gyri of man and apes, but of their paracentral lobule.

After a short account of the sensory division of the brain, which is only too meagre in detail, our author sketches lightly the resemblance betwixt the rough conformation of the cerebral hemispheres and the spinal cord as regards its bilateral,

motor, and sensory segments, its commissural connections, &c. Referring to the peculiar site of the "giant cells," which he regards as motor, and their relationships to the great venous sinus, and large veins, he suggests this as a plausible explanation of many cases of transitory paralysis of centric origin, of puerperal eclampsia, epileptic spasm, hysterical laughter, partial and complete aphasia.

Prior to the appearance of this memoir by Betz, these great cells had been noted by several observers, and were regarded as "hypertrophied nerve-cells;" but to Betz is due the credit of indicating their constant presence in healthy brain, and their definite localisation in the paracentral lobule. In claiming for them a motor significance, however, he simply followed out the idea expressed years previously by Meynert,[1] that the cells of the third layer closely resemble those of the anterior cornua of the cord, and the nuclei of the motor cerebral nerves. The points to which objection must be taken in the above article refer to—

a. The distinctive appellation of "giant cell."
b. The position of these cells in the cortical layers.
c. Their distribution over the superficies.
d. Statements regarding the gyri of carnivora.

I have elsewhere[2] stated my conviction, together with that of a fellow-worker, that the term "giant pyramid" is inapplicable to these cells, since they were but great representatives of a very general formation varying greatly in size. In the article referred to we stated that "the disparity in size between these elements in the fourth layer of the motor area induces us to reject the term 'giant cell' as applied *generally* to this formation, and to call them by preference the ganglionic cells of the cortex."[3] It was shown by us that these ganglionic cells were not only extremely varied in contour, and not by any means constantly pyramidal, but that they were represented over a much greater area of the brain than was affirmed by Betz—in fact, were spread over the whole vault where in

[1] 'The Brain of Mammals,' by Th. Meynert. Stricker's 'Human and Comp. Histology,' Sydenham Soc. vol. ii. p. 387.

[2] "Cortical Lamination of the Motor Area of the Brain." Proceedings of the Royal Society, No. 185, 1878.

[3] Op. cit.

most positions their dimensions did not exceed the lower cells of the third layer. From these facts it will be seen that the term used by Betz is inadmissible.

Again, as regards their position in depth, it was shown that these peculiar cells could not be regarded as invariably situated in the *fourth cortical layer,* as over the greater area of the vault the cortex was distinctly *six-laminated,* and these cells consequently would here occupy the *fifth layer.* It was shown that Betz [1] and Mierzejewski,[2] following Meynert,[3] were wrong in regarding the general type of cortical lamination as five-layered, whilst Baillarger was correct in representing it as six-laminated. The true position is, as we first indicated, that there was both a five and a six-laminated cortex, the former specially characteristic of Ferrier's motor area, the latter of sensory realms where the interposition of a granule belt formed a six-laminated cortex.

CORTICAL LAMINATION OF VAULT.

	After Meynert, Betz, and Mierzejewski.	Baillarger.	Lewis and Clarke.	
			Motor Sensory.	Motor Sensory.
1st Layer .	The Neuroglia zone.	Do.	Do.	Do.
2nd „	„ Small pyramidal cells.	Do.	Do.	Do.
3rd „	„ Large pyramidal cells.	Do.	Do.	Do.
4th „	„ Irregular granule cells.	Do.	Ganglionic cells.	Granules.
5th „	„ Spindle cells.	Large pyramids.	Spindle cells.	Ganglionic.
6th „		Spindle cells		Spindles.

The position held by us is one of importance, since by it the sensory realms are shown to acquire a new layer by the intercalation of a belt of granule cells *above* the ganglionic formation. Later on, in a memoir published in 'BRAIN,' it was shown that the belt of granule cells was richly developed in proportion to the fading off of the ganglionic series, and *vice versâ.* Whilst fully admitting their nested arrangement and other special features described by Betz, we wholly dissented

[1] Op. cit.

[2] 'Archives de Physiologie,' 1875. "Études sur les lésions cérébrales dans la paralysie générale."

[3] Op. cit.

from their limitation to the narrow districts which he assigned them. Exception must also be taken to his statements concerning the analogue of the central gyri in carnivora. Professor Betz concludes, from a consideration of the minute structure and relationships of the paracentral lobule in man and the higher apes that its analogue is to be found in the sigmoid gyrus, and that the latter does not represent the central gyri of higher animals and man—"the central gyri of dogs being wholly absent." This latter assertion is quite unwarranted, since, by a far more philosophical method of inquiry, Broca has shown that the central gyri, separated by a well-marked sulcus (Rolando),[1] are reproduced at the frontal pole of the brain in carnivora. According to Betz, the frontal end anterior to the "motor region" is the representative of the frontal gyri, and no true Sylvian fossa is shown. The former statement is in every way inaccurate; and as to the latter, a Sylvian depression is certainly seen.

In his later memoir our author states that hitherto neither the researches of Fritsch and Hitzig nor those of Ferrier have been afforded a basis in the anatomical construction of the brain, and advances the results of his recent investigations as supplying the deficiency; thus totally ignoring the classical works of Charcot and of Meynert, which most strongly point in this direction; as also my own investigations on the cortex cerebri, published between the years 1878 and 1880. Preparatory to giving the details of his work he describes the general laminar type so as more clearly to establish the divergencies which occur. He again reiterates his earlier statement that this type is *five*-laminated, but his description or summary is both defective in detail and exactitude, and cannot be fairly contrasted with the descriptions afforded by other authorities, Meynert, &c. &c. Thus in alluding to the first layer he speaks of it as simply an extension of neuroglia, totally ignoring its complicated nervous network derived from the subjacent nerve-cells, as well as its peripheral medullated zone and corpuscular elements. Then follows a summary of the varieties of cortical structure, based upon the examination of 5000 preparations

[1] "Anatomie Comparée des Circonvolutions," par Paul Broca. 'Revue d'Anthropologie,' 1878.

from the right and left hemispheres of the same individual; also from the brain of male and female, young, adult and aged subjects; as well as in the brains of a seven months' fœtus, a new-born child and an infant six weeks old.

The whole cortex cerebri, which he previously divided into a motor and sensory portion, is now subdivided by him into the following realms, differing from one another in histological constitution :—

1. Ascending frontal gyrus.
2. Gyrus fornicatus.
3. Cornu ammonis.
4. Third or inferior frontal gyrus.
5. Paracentral lobule.
6. Lingual gyrus.
7. Tip of the temporo-sphenoidal lobe.

Let us contrast with this enumeration Meynert's five types of cortical lamination, which are as follows :—[1]

1. Type common to vault and gyrus fornicatus.
2. Occipital apex.
3. Sylvian fissure.
4. Cornu ammonis.
5. Bulbus olfactorius.

We here see that Betz has subdivided Meynert's first district into three subdivisions, whilst his lingual gyrus really embraces the same type of structure as Meynert's occipital type. The object of Meynert's enumeration was to present us with generic divergencies in structure, and this he has faithfully done; for all his types, with one exception, allude to cortex which differs in kind rather than in degree, in the actual absence of certain layers or addition of new layers. Betz, however, appears to dwell chiefly upon the more minute specific differences in the layers and their elements, and with this object has dwelt upon each individual convolution; yet in his summary, as given above, he has inextricably confused the fundamental distinction to be always kept in mind, between areas whose structures vary in kind or in degree; and thus such wholly different structures as the cornu ammonis, lingual gyrus, tip of temporal pole, are

[1] Op. cit. p. 381 (published 1872).

mixed up with the various regions which he regards as five-laminated. We would insist strongly upon the necessity of upholding the above distinction, otherwise results given by individual inquirers must be confusing and conflicting. Nor do we see why he should exclude (upon his own principle) the olfactory bulb from his tabulation, as it is as surely a portion of the cerebral cortex as the cornu. Space forbids a detailed account of each individual gyrus as given by Betz, but as regards the site of the "giant pyramids," it is now stated that typical nests of these great cells are found in the paracentral lobule, and extend over the upper end of the ascending frontal gyrus—deep in the fissure of Rolando at its median extremity and very exceptionally on the anterior surface of the ascending parietal. It is here stated that giant cells of "small calibre" are found grouped at the origin of the two upper frontal gyri—also at the posterior end of the lowest frontal, extending back into the ascending frontal. This localisation by Professor Betz confirms in a remarkable way the results published by the reviewer two years ago.[1] A critical survey of the divergencies in structure of the cortex in the various gyri unfortunately affords us very little additional information. The peculiarities in structure of the lingual gyrus, of the temporo-sphenoidal lobe, and of the olfactory bulb were all known and long since described by Meynert, and the more trivial differences in most of the other gyri (with exception of the more special motor realms) simply obey the law expressed by the writer some years since, as follows: "No *abrupt* passage from one form of cortical lamination to that of another is ever seen in the convolutions of the vault. Changes in type are gradually assumed, and no distinct boundary exists to indicate an exact line of demarcation. The structure peculiar to one region gradually fades and blends with arrangements which foreshadow the architecture of adjacent realms, and thus we obtain *transition regions*."[2] It is in the enumeration of such transition regions that Professor Betz chiefly engages when

[1] Loc. cit.

[2] "Comparative Structure of the Cortex Cerebri;" Bevan Lewis, in 'Brain,' Part I. 1878, p. 84. 'Proc. Roy. Soc.' No. 185, 1878; and in 'Brain,' Part I. p. 83.

not describing the really typical varieties of the cortex. Again, in his very cursory examination of the structure of the insula, the features of this important region are given in a most vague and inexact manner, nor is any reference made to the very thorough examination of its lamination which was described by Dr. Major in 1876.[1] It is much to be regretted that in this later memoir the same scientific exactness does not appear which characterised the former article and which was its especial charm. Our author had apparently entirely discarded the use of the micrometer, and the vague expressions, larger, smaller, thick, or thinner, are constantly employed; and his results are therefore to a great extent removed from the test of comparison with those of other inquirers. This is all the more to be regretted, as the special portion of his work which is stamped by greater exactness, viz. that of the localisation of the nested "giant cells," is so substantially a confirmation of what had previously been advanced. There are other points of importance upon which we must join issue with our author. In his former memoir he describes the "giant cells" as specially localised in the paracentral lobule and upper end of the ascending frontal; in the later memoir he extends their limits, and refers to these distant elements as "giant cells of small calibre,"—surely a most inappropriate term when these elements resemble in size the cells of the lowest stratum of the third layer. We are safer in terming them ganglionic cells, and in recognising their existence over the greater portion of the vault, and especially decreasing in size towards the lower end of the ascending frontal (*vide* Measurements).[2] Again, we must strongly insist upon the point that the *general type* of cortical lamination is of six, and not five layers; and would suggest as the cause of conflicting opinion on this point the neglect of fresh examinations of the cortex.

The method adopted of distinguishing the individual layers by number is another element of fallacy; and far less confusion would be introduced if each layer was named after its prominent morphological character. Thus I am in the habit

[1] 'Histology of the Island of Reil.' Dr. Major, 'West Riding Reports, 1876.'

[2] "Comparative Structure of the Cortex Cerebri;" Bevan Lewis, in 'BRAIN,' Part I. 1878, p. 84. 'Proc. Roy. Soc.' No. 185, 1878; and in 'BRAIN,' Part I. p. 83.

of describing the *general cortical type* as consisting of the following layers:—

1. Peripheral cortical zone.
2. Small pyramidal layer.
3. Large pyramidal layer.
4. Small angular or granule layer.
5. Ganglionic layer.
6. Spindle layer.

W. BEVAN LEWIS.

Bernhardt on Cerebral Tumours. (Beiträge zur Symptomatologie und Diagnostik der Hirngeschwülste.) Von DR. M. BERNHARDT. Berlin, 1881. 8°. pp. 336.

THIS work is a laborious abstract and tabulation of the symptoms which have been recorded by different observers in connection with tumours situated in various parts of the encephalon. The list is extensive, if not complete, and though the cases are not reported with such a degree of fulness as to dispense with the necessity of referring to the originals, yet clinical workers will be thankful to Dr. Bernhardt for saving them from much tedious labour. Beyond the valuable collection of facts, there is little calling for special remark. Bernhardt's work has been anticipated, and we think greatly surpassed in lucid arrangement and treatment, in the chapters devoted to cerebral tumours in Nothnagel's treatise on the Regional Diagnosis of Cerebral Disease.

D. FERRIER.

Clinical Cases.

CASE OF BRAIN LESION WITH HEMIPLEGIA ON THE SAME SIDE AND UNI-OCULAR NEURITIS ON THE OPPOSITE SIDE.

BY ERNEST FIELD, M.D., BATH.

H. R., æt. 26, first came under my care in June 1879, with the following history :—Five or six years before he contracted syphilis, but with that exception had enjoyed good health until June 1876, when he had a slight attack of hemiplegia on the right side. This occurred during the night, and was only noticed by him on waking in the morning. It was, however, not enough to confine him to bed beyond a week, and at the expiration of that time he was able to walk, with assistance, to the hospital, where he remained as an in-patient for a week or two.

He recovered sufficiently to return to his work, at which he continued more or less up to May 1879, though his intellect became much duller, and he suffered throughout from some weakness of the limbs of the right side.

His condition when he came under my care, was as follows :—Distinct, though slight, loss of power in the right arm, with a little dragging of the corresponding leg; no loss of sensation on this side, but in the left thigh sensation distinctly diminished, and in it only; some drooping of the left eyelid, with complaint of occasional diplopia.

On both legs were the scars of old rupia.

He lay in bed in a peculiarly lethargic, somnolent, condition, with scarce sufficient power to raise himself, and from time to time had retention of urine, for which the catheter was necessary; fæces occasionally passed involuntarily, but only after the administration of purgative medicine, which the obstinate constipation from which he suffered rendered necessary.

Eyes normal, beyond irregular dilatation of pupils on introduction of atropine, the result of part iritis.

He was ordered perchloride of mercury, iodide of potassium,

and tonics, and under this treatment he slowly improved and was again able to get about; but during the next twelve months he was subject to these attacks of lethargy and general loss of nerve power, removed by a return to specific remedies.

For the four months preceding his death he was able to walk down to the dispensary, a distance of half a mile, and he did this up to two days before his fatal seizure. During this last period he had an attack of optic neuritis, confined to the left eye, with hæmorrhages on and around the disc, but from this he completely recovered, with good vision.

An examination of the knee-reflex about two months before death showed it to be present in an exaggerated form in the right leg, but absent in the left. No clonus at the ankle was obtainable on either side, but there was great resistance to dorsal flexion of the foot.

Epileptiform attacks were present during the last year of life, but I was never able to see him in one. On the morning of his death he was found in bed in a comatose state, breathing stertorous and irregular, pupils dilated but equal, pulse 140; and after a few hours he died.

The diminution in sensation noticed in the left thigh had quite disappeared for some months before death, and the only paralysis existing was the slight affection of the right side before described.

Pain in the head was not a prominent feature at any time, though on being questioned as to whether he had any, he usually replied in the affirmative.

At the *post-mortem* examination I had the kind assistance of my friend Dr. Berry, at that time house-surgeon to the Eastern Dispensary.

On removing the calvarium the contents seemed tightly packed and the vessels somewhat engorged. In the ventricles and spinal canal was a large quantity of cerebro-spinal fluid. At one spot on the right side, in the situation of the operculum, the dura mater was firmly adherent to a hard substance at the surface, occupying an area about the size of a half-crown piece; but the whole hemisphere around this was much softened—in fact, quite diffluent, only the occipital lobe and the extreme anterior part of the frontal lobe were normal in consistence. This extreme and widely-spread softening rendered exact localisation of the tumour somewhat difficult. The growth was of a slaty appearance on section, and reached the surface in the region of the operculum; of irregular, nodulated shape, it was in all about the size of a pigeon's egg.

The left hemisphere was fairly normal. In the ependyma covering the corpus striatum was a small, yellowish thickening at one spot, the area of a shirt-button. Neither corpus nor

thalamus was quite as plump and large as usual, but there was nothing very marked wrong with them.

On microscopic examination the tumour was found to be composed of small round cells, with a delicate reticulum, resembling imperfectly developed adenoid tissue. In parts much caseous change had occurred.

Remarks.—There are two features of special interest in this case, viz. the fact that the hemiplegia and the brain lesion were on the same side, and the occurrence of uni-ocular optic neuritis.

As is well known, Flechsig has pointed out that there is great variation in the amount of decussation which takes place in the anterior pyramids ; whereas usually the larger portion of fibres cross over to the opposite side of the cord, passing down in the posterior part of the lateral column, whilst the smaller portion only remains on the same side, constituting the anterior median column, yet occasionally this is not the case, and the mass of them remain on the same side. Whether any such arrangement obtained in the present case I am unable to say, but without it there is much difficulty in explaining the phenomena observed.

The usual difficulties attending autopsies in private houses deterred me from removing the cord; but it is much to be regretted that this was not done, as the increased knee-reflex on the affected side rendered the existence of some descending degeneration probable, whilst the position of the latter might have thrown some light on the decussation which had occurred in the anterior pyramids. For had we found the mass of the degeneration in the anterior medium column of the right side, the posterior part of the lateral being intact, whilst on the left side a small portion only of the lateral column was diseased, it would have amounted to proof that the ordinary decussation of the anterior pyramidal fibres had not taken place.

With regard to the optic neuritis, it is noticeable that it was situated on the side opposite to the tumour, a condition observed by Dr. Hughlings-Jackson in two cases of unilateral neuritis recorded by him. And this fact would certainly appear to lend weight to the hypothesis advanced by Dr. Stephen Mackenzie, that the occurrence of optic neuritis in connection with brain disease is due rather to a diffuse cerebritis continued down the nerve than to pressure.

Another remarkable point to notice is that, notwithstanding the continued presence of a fatal lesion, the eye should recover itself and good vision return. Recovery from optic neuritis with good vision is, of course, frequently seen, but only in cases where the existing cause can be removed.

ON A CASE OF TUMOUR OF THE CEREBELLUM WITH LEFT HEMIPLEGIA.

BY CHARLES E. BEEVOR, M.B.

Resident Medical Officer to the National Hospital for the Paralysed and Epileptic, Queen Square.

THE following case was under the care of Dr. Ramskill, at the National Hospital for the Paralysed and Epileptic, Queen Square, and it is through his kindness that I am able to publish it.

F. D., a boy aged 11, was admitted into the hospital on June 22nd, 1880, and the following history was obtained from his mother.

There was no neurotic history, and he had had no previous illness of any importance.

Present illness came on five months ago rather suddenly, with pains in the head, and vomiting after everything he took. Four months ago he had a fit while in bed, and preceded by vomiting; he did not lose consciousness; the face was "drawn down on the right side," but the arms and legs were not convulsed; the face was not paralysed after the fit; he had another similar fit a week later.

About this time his sight began to be impaired, and in about another fortnight his mother noticed that his eyes were turned inwards, and they have remained so ever since. Two months ago he began to get giddy, and to reel about in his walking, and to fall forwards and to the left, and "one day he lost power" in the left arm and leg, but not completely, as he was able to walk with assistance till within a month of his admission; the left arm and leg have been getting gradually worse ever since.

His mother does not think that the face was affected the same time as the left arm and leg; but it probably was, as it was found to be affected on the left side on admission. His mother also states that he has not been able to lie on his left side for the last three months.

Present state.—Patient is a dull, heavy-looking boy, who does not take notice of anything, but answers questions slowly,

and takes a long time to do what he is told. He is very drowsy, and sleeps very much, and does not like to be disturbed.

He says he has not any pain at present, but frequently has severe pain in his forehead.

He has frequent vomiting, and the absence of food makes no difference, or very little.

Face.—On showing the upper teeth the right side of upper lip moves, and the left hardly at all; he can close both eyes equally well. Has no ptosis.

Ocular muscles.—There is internal strabismus of the right eye; he can direct either eye to the inner side, but cannot turn either eye outwards beyond the middle line; he cannot elevate or depress the eyes at all; he apparently cannot converge the eyes to a greater degree than they are already converged (?). Pupils equal, rather dilated, no reaction to light, contraction to accommodation doubtful.

Muscles of mastication not affected, and he protrudes the tongue a little to the left.

In phonation the soft palate seems to be drawn a little more to the right than left.

He swallows slowly and with difficulty.

Upper limbs.—Both wasted. Left measures about $\frac{1}{4}$ inch less than right.

Grasp of right hand = 2 kilogrammes by dynamometer.
" left = 0 " "

Has free movement of right arm.

With the left he cannot pick up a pin; he can just manage to raise the left hand to his head, but cannot hold it out straight in front of him; can just flex and extend the left fingers. He is unable to raise the left shoulder.

Lower limbs are much wasted, especially the left. He has free movement of the right leg, but he cannot move the left one at all, and it falls like a dead weight. No rigidity in either limb.

The power of right leg is much below normal, but this is probably due to his general weak condition.

Knee phenomenon obtained in either leg, but no excess can be made out.

Ankle clonus present in left leg, but not very well marked; absent in the right.

Plantar reflex present on both sides, but more marked on the right than the left.

Cremasteric reflex very well marked on the right side, not obtained on the left.

Abdominal and *Epigastric* reflexes very marked on the right, only just obtained on the left.

Sensibility does not seem to be affected, and he localises correctly where he is touched, on both sides all over the body.

Sight.—No diplopia; he can recognise objects—as a key—but cannot distinguish letters.

Ophthalmoscopic examination shows intense double optic neuritis, both discs very red and the margins obscured.

Hearing is much affected, a watch being heard about 12 inches away with the right ear, and only 2 inches off with the left ear.

Taste and *Smell* could not be properly ascertained, owing to the dense condition of his intellect.

Urine contains no albumen. He passes urine and fæces involuntarily into the bed.

He has almost constant priapism.

There is nothing abnormal in the heart, pulse 64.

The vomiting persisting three or four times a day, he was fed for a time on nutrient enemata only, but he still vomited every morning.

On July 15th he had a fit; he lost consciousness, and the head was drawn round to the left; there was marked arching of the back; the arms and legs were not convulsed, but became very stiff, especially the legs; the attack lasted about one minute, and in a few minutes he had another of a similar nature. He was very drowsy after them.

Patient gradually got worse and more dense and drowsy, and as far as could be ascertained he did not present any fresh symptoms; the vomiting persisted, and though he was fed with nutrient enemata he gradually sank, and died on August 4th.

His temperature on the evening before he died was 104, and just before death was 107·4.

On post-mortem examination a tumour was found shaped like a cottage loaf, measuring 3 inches long, and 2½ broad at its larger end, by which it grew from the upper surface of the cerebellum, extending on each side of its middle line. The smaller end of the tumour was free, and extended forwards between the optic thalami, and flattening the corpora quadrigemina, so that there was hardly any trace of them visible; the right optic thalamus for about the inner ¼ of an inch, the right crus cerebri, and the right superior cerebellar peduncle, were also compressed; there was some thickening of the meninges about the tumour, but there was no sign of pressure on the pons, medulla, or any of the cranial nerves.

In this case there were the following symptoms met with in intra-cranial tumours: pain in the head, daily purposeless vomiting, and double optic neuritis.

The localisation of the tumour to the cerebellum was not warranted by the symptoms as they first presented themselves,

as during the first month he only had pain in the head and constant vomiting. The description of the fits as given by his mother was very imperfect, and she did not know very much about them, but those he had in the hospital were cerebellar in character, arching of the back with tonic rigidity of the legs, and absence of clonic convulsions. The first fit occurred about a month after onset, and about this time his sight began to fail; but whether this was due to the optic neuritis is doubtful, as Dr. Hughlings-Jackson has shown that double optic neuritis may exist and yet with no impairment of vision.

In one and a half to two months after onset of illness he was one day noticed to have internal strabismus, and he gradually became giddy and staggering in his walking, falling forwards and to the left, and he suddenly one day lost some power in the left arm and leg.

It seems probable that the tumour, which had hitherto kept to the middle line of the cerebellum and had produced no symptoms pointing to its position, now began to grow forwards and to the right, and press on the right crus cerebri; for it has been found that lesions of the cerebellum *per se* do not cause hemiplegia. The loss of power in the limbs was not complete, but gradually increased as the pressure of the growth on the crus became more and more; the leg being affected, on admission, more than the arm, and so differing from ordinary cases of hemiplegia, and thus bearing out Dr. Bastian's opinion (Paralysis from Brain Disease) that these cases are due often to pressure on the pons Varolii from lesions of the cerebellum.

With regard to the strabismus and paralysis of the movements of the eyeballs, they were probably due to the compression of the corpora quadrigemina, which are now considered to be the centres for the co-ordination of the movements of the eyeballs; for in this case there was no paralysis due to lesion of any one nerve, for the portions of the third nerves supplying the internal recti were not affected, while the parts of the nerves supplying the superior and inferior recti were paralysed, clearly showing that it was not the nuclei or the nerve trunks themselves that were affected, but a centre higher up, which co-ordinates the movements of the eyes in different directions; and this case would seem to bear out the theory that the centres for converging the eyes, and for elevating and depressing them were paralysed, while the centre for causing lateral movement was very much impaired.

The corpora quadrigemina are not now considered the seat of vision, and in this case, though they were so much com-

pressed, the patient was able to see well enough to distinguish objects.

The pupils were immovable, and this has been found in other cases to be associated with bilateral lesion of the corpora quadrigemina.

With regard to the situation of the tumour in the median lobe of the cerebellum, there was one symptom which Dr. Bastian (Paralysis from Brain Disease) has stated to be met with most commonly in lesion of this part, viz., persistent priapism, which occurred the whole time he was in the hospital.

The superficial reflexes in this case were altered as they frequently are in hemiplegia, viz., diminished on the paralysed side; the plantar, abdominal and epigastric reflexes being less marked on the left than on the right, while the left cremasteric was absent, the right being very active; on the other hand, ankle clonus was obtained on the left side, but absent on the right—no difference could be made out between the knee phenomena of the two sides.

This diminution of the superficial reflexes on the paralysed side has been explained by a theory based on Setschenow's experiment, who stimulated the optic lobes in a frog and thereby found that reflex action was not so active, the optic lobes exercising an inhibitory action over the reflexes. In man it has been supposed that the optic thalami exercise this inhibitory power over the superficial reflexes; but normally the thalami are themselves inhibited by the cerebral hemispheres, so that when hemiplegia occurs from a lesion cutting the connection between the hemisphere and the optic thalamus, the latter has full play and inhibits the superficial reflexes of the paralysed side.

In this case, however, the superficial reflexes were diminished on the paralysed side, the connection between the optic thalamus and the hemisphere was intact, and the lesion was situated below the optic thalamus and between it and the spinal cord, so, in the first place, the optic thalamus was still inhibited by the hemisphere; and, secondly, the spinal centres were cut off from the influence of the thalamus.

Besides, if this theory were a correct explanation, the superficial reflexes ought to be diminished directly the inhibitory action of the hemisphere is removed—as by a sudden hemorrhage—and at the onset of the attack; but this is not found to be the case, the superficial reflexes not being absent or diminished on the paralysed side till some time after an apoplectic seizure.

The staggering in his walking and the falling forwards and towards the left side, which occurred about six weeks or two

months after the onset of his illness, were probably due to pressure of the tumour on the right half of the cerebellum, and on the right superior cerebellar peduncle.

The right half of the cerebellum works with the left half of the cerebral hemispheres, and they both govern the right half of the body, and when one half of the cerebellum is pressed upon by a tumour, the opposite half over compensates and carries the body over to the opposite side (Dr. Ferrier), and in this case, with a lesion of the right half of the cerebellum, the body was carried over to the left side. Falling forwards has also been considered as occurring in lesions of the anterior part of the median lobe of the cerebellum.

The power of hearing was very much affected, and on the left side more than on the right, which might have been caused by the pressure on the right crus cerebri involving the fibres passing up from the auditory nucleus to the higher centres for hearing in the cerebral convolutions. Hearing on the right side being affected would probably be due to the deficient condition of the patient's intellect; but, besides this, the theory has been advanced as to the possibility of the auditory nerves suffering from neuritis, from intracranial pressure, in a manner similar to the optic nerves, and thus their functions being impaired.

The involuntary evacuation of excreta—which is rare in young cases of hemiplegia—could be accounted for by the patient's mental condition, as he did not take notice of anything, and only spoke when asked a question.

For the appended account of the microscopical appearances of the tumour I am very much indebted to Mr. Victor Horsley, assistant pathologist at University College, who very kindly examined the growth for me.

Tumour of Cerebellum.

The growth in this case evidently sprang from the under surface of the dura mater, extending inwards towards the brain. Throughout it was soft and greyish in colour, obviously cystic in the older portions and more uniform in texture towards the growing edge. Microscopical examination showed it to consist of a fibrous stroma and two classes of corpuscular elements.

The stroma growing from the dura mater was composed of dense fibrous tissue at the periphery, but soon divided into more delicate trabeculæ consisting of fibres and spindle-cells, while here and there patches of small round cells, forming a loose embryonic tissue, were found in the more actively growing portions. In several places the stroma was evidently

myxomatous, while everywhere the fibrous trabeculæ formed alveoli containing the round cells described below, and sometimes presenting even the appearance of an alveolar sarcoma. Besides the connective tissue corpuscles of the stroma, there were two distinct classes of cells which formed the main mass of the tumour.

1. Round corpuscles. These were large round or oval cells with granular protoplasm, a large nucleus, and bright nucleolus, and measuring about $\frac{1}{1000}$th inch in diameter. These occupied the alveolar spaces in the stroma sometimes in large masses, at others the cells would be enclosed in little single meshes.

2. The second class of corpuscles constituted an epithelial growth extending through the tumour in different directions. In its earliest stage it consisted of columns of cells which at the periphery were columnar in shape, but towards the centre of the column passed through the transitional stages until the central cells were large flat epithelial plates (figured by Cornil and Ranvier, part 1, p. 134). In other cases the cells in the centre of the column had undergone extensive colloidal change, and this had evidently led to the production of the cysts described below. The epithelial corpuscles were also scattered in groups through the tissue of the stroma without apparently any columnar arrangement, and were perfectly distinct from the large round cells occupying the alveoli of the stroma, since the latter spaces were bounded by well-marked trabeculæ, while the epithelial columns were always found where the stroma was myxomatous.

An additional point of interest in the tumour was the formation of cysts. These cysts were of various sizes up to 2 mm. in diameter, and situated in the older portions of the growth, containing either simply granular débris, colloid material or cells which were not completely degenerated.

No relationship could be detected between the blood-vessels and epithelial columns, and the groups of epithelial cells had not become calcified.

The growth is obviously that described by Cornil and Rauvier under the name of "Angiolithic sarcoma," by Virchow as "psammoma," and Rindfleisch as "Epithelioma myxomatodes psammosum." Neither Cornil and Ranvier nor Rindfleisch mention a round-celled growth which was so striking a feature in this case.

The spinal cord on examination proved to be healthy throughout.

SPASMODIC WRY-NECK; EXCISION OF A PORTION OF THE SPINAL ACCESSORY NERVE—DEATH FROM ERYSIPELAS.

BY WALTER RIVINGTON, F.R.C.S.

Surgeon to the London Hospital.

HENRY H., 58, was admitted into the London Hospital in February 1875, under Dr. Hughlings-Jackson. The notes of the case were taken by Mr. Charles Mercier.

The patient had the ordinary form of spasmodic wry-neck, and had suffered about eighteen months. He was a healthy-looking man, and said that he had had no serious illness except small-pox when a child. He never had rheumatic fever or scarlet fever. He did not know that any of his blood-relatives had suffered from any kind of nervous affection. Detailed inquiries were made in this regard, and his statement must of course be taken for what it is worth. Some years ago the patient, who had been a weaver, suffered from what we may call "weavers' cramp," by which is meant a condition analogous to "writers' cramp." The following account of it was obtained by Mr. Richard Atkinson, on the patient's second admission, but is conveniently narrated here as part of his history. It was no part of the case, in so far as association with the wry-neck goes, but it may possibly be supposed by some to show a neurotic tendency: "In weaving, the operator throws the shuttle, to which the warp is attached, through the woof, and then catches it with the other hand. He then performs the same motion in the reverse order, so that the shuttle is brought back to the hand from which it started. In throwing the shuttle it is held between the thumb and middle finger, the index-finger resting on it behind. The propulsion is effected by a slight flexion of the wrist together with a flexion of the index-finger. The patient performed exactly the same motions with his right hand as with his left, but he never got the cramp in his right hand, only in his left. The cramp came on thus: Having thrown the shuttle from

his left hand, he would carry the wrist backward so as to be ready to catch it again when returned from the right hand, and it was after throwing the shuttle that the cramp seized his hand so as to overflex the fingers and prevent him being ready to catch the shuttle on its return from his right hand. Great accuracy and rapidity of movement being requisite for weaving purposes, the patient was thus on several occasions unable to follow his trade. He took to another and simpler method, and for some years he succeeded in this; but again the cramp seized him and, as before, the left was the hand affected. In this method he used his left hand only to push a beam, called a batten, backwards and forwards. The right hand threw the shuttle through the woof; instead of the left hand catching and returning it there is a box which does this. The movement seems to have consisted of an alternate slight flexion and extension of the elbow, the wrist being kept a little extended. Though there would be little or no movement of flexion and extension performed by the wrist, yet there would be considerable strain on the flexors of the fingers. In this method of weaving, as in the previously described, the cramp consisted of an over-flexion of the left wrist, so that he became unable properly to remove his batten." The following is Mr. Mercier's note of the case in February 1875: "He first noticed the onset of this complaint eighteen months ago. At that time he had an aching pain at the sides of his neck (he indicates the upper portion of the sterno-mastoids), equally bad on both sides. It was just as if he had sat in a draught and caught cold. It was not persistent: was worse when he turned his head in either direction, and went off if he kept his head for some time still in the middle line. It was always absent at night. Afterwards he had a pain at the back of the head on a level with the occiput, on the right side only, an aching pain. This pain has never been on the left side. He had had the pain for six months before the head began to move. At first the movement was very slight indeed; when he imitates it, it is scarcely perceptible, and as he reproduces it, it is a simple rotation from left to right, without any lateral or oblique movement. He could get it back by effort, but it immediately returned, not more strongly, however, for the check it had received. At this early date it never twisted the head round even after repeated spasms. It very gradually got worse and worse, with an occasional remission for a day or two. At first the spasm ceased directly he put his head on the pillow, but gradually he began to have it at this time also. His wife does not know whether he moves his head during sleep. He says that his head is moving when he wakes in the morning. He has always had pain like a cramp in the back

of the neck whenever he moves his head round much. At the present time the movements are usually a slight rotation of the head towards the right, while at the same time it leans down towards the right shoulder, and the chin is raised. During the movement the left sterno-mastoid becomes extremely hard, and the left trapezius hardens slightly. At times the rotation is very much greater, and the head leans much more over the shoulder, the chin is also raised more, but not so much more. In these more extensive movements the left sterno-mastoid is quite rigid, and the left trapezius is also brought strongly into action. In the return movements, the corresponding muscles on the opposite side contract. The movements consist of a rapid succession of small ones scarcely abrupt enough to be called jerks, but yet distinctly separate."

November 1st, 1875.—Patient has been attending on and off ever since. He wore an apparatus with benefit for some time, but the torsion has become so bad that it is no longer useful. He is thinner, and has done no work for twelve months. He was re-admitted on January 21st, 1876.

No kind of treatment did the man any good excepting the temporary benefit from the apparatus. Dr. Hughlings-Jackson consulted Mr. Rivington as to the propriety of dividing the spinal accessory nerve, and, after consultation, advised the patient to have the operation performed. He was transferred to Mr. Rivington's care, whose further report concludes the case.

"The patient was a man of slender build with a feeble circulation. The heart-sounds were normal but weak; breath-sounds were feeble, expiration being prolonged. Urine was free from albumen.

"The operation was performed on February 7th, after the method adopted by Mr. Campbell de Morgan. The patient having been anæsthetised, an incision was made along the posterior border of the left sterno-mastoid for 2 or 3 inches. The skin superficial fascia and platysma were divided successively. The external jugular vein, which was in relief from distension with blood, was carefully protected and drawn aside. The deep cervical fascia was then cautiously incised, and the great auricular nerve was recognised ascending towards the lobule of the ear, and near this the spinal accessory was found. The nerve was then traced through the substance of the sterno-mastoid, and a piece was excised from the trunk before its entrance into the muscle. Galvanism was employed to verify the nerve, the sterno-mastoid being thrown into strong contraction when the lower part of the divided nerve was included in the circuit. One small vessel was ligatured, and there was some oozing during the performance of the operation. The

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edges of the wound were brought together with wire sutures, pads of lint and strapping being applied, and a bandage over all. After the effects of the chloroform had passed off, the neck was examined. The spasms and twisting of the neck still continued. These were found to be caused not by the left sterno-mastoid, which was quite relaxed, but by the action of the muscles at the back of the neck on the right side. The right splenius was tense. The head was twisted quite as much as before the operation, the face being turned round to the right side, and the chin tilted upwards. As it was evident that nothing more could be done, the patient was sent to bed. He had a dose of chloral hydrate in the evening and slept for some hours. Early the next morning the patient shivered a good deal. His tongue was dry and furred. Pulse was 120, moderately strong, and the temperature stood at 103°·2. The spasms continued intermittently.

"*Feb. 9th.*—Patient slept again last night pretty well after chloral hydrate. Tongue slightly furred. Appetite fair. Bowels inactive. Pulse 90 and moderately strong. Skin moist. Wound clean. Wound dressed by Mr. Mears (the dresser to the case, from whose notes most of these particulars are extracted). Wound looking clean; very little discharge; no tendency to bag; edges seem united save at one spot. Wet lint applied. Movement of head nearly stopped; scarcely if any jerking.

"*10th.*—Wound healthy; edges united in parts; highest stitches taken out; no bagging, pulse 84, temp. 99°·2. Head quiet.

"*11th.*—Head still quiet; bowels inactive; tongue slightly furred; appetite moderate, pulse 98·2. Wound healthy; patient slept well after chloral hydrate.

"*12th.*—Patient says he is better; tongue cleaner; bowels have acted. Pulse 84, good, temp. 98°·2. Remaining stitches removed; wound united in the middle; head quieter.

"*14th.*—Not much sleep last night on account of pain at back of head. Appetite good. Pulse 90, fairly strong; temp. 99°. Wound very healthy, moderate discharge of healthy pus, granulating rapidly.

"*16th.*—Temperature 103°·2 in the morning, no apparent cause for the rise. Patient otherwise in good condition.

"*17th.*—Discharge slight, health good; temp. 193°. Head quiet.

"*18th.*—Wound not quite so healthy, skin dry, scaly and purplish, no erysipelatous tint; secretion of a dirty brown colour and scanty. Patient sleeps well and has a fair appetite, temp. 98°·4; pulse regular, 96; bowels active.

"*19th.*—Patient restless all night in spite of hyd. chlor.

draught. Tongue dry and dark; pulse 108, sharp and rather weaker; skin moist; temp. 100°·2.

"21*st.*—Erysipelas appeared yesterday, temp. 103°; pulse 112; lot. plumbi evaporans ordered. To-day the erysipelas has spread over the scalp, left cheek, neck and shoulder. Temp. 104°; pulse 102, full; tongue brown and dry, skin hot and moist; no appetite; dirty pus scantily secreted.

"22*nd.*—Patient slept a little. Removed to Erysipelas ward, and ordered quin. sulph. and fer. perchlor. Skin moist; temp. 103°; no secretion from wound, but a yellow crust on the surface. Erysipelas has spread right over the scalp, left cheek and eye, left side of neck, and upper part of thorax; in some places blebs have formed.

"23*rd.*—Erysipelas has invaded opposite side of neck and has become deeper seated; bulloe everywhere. Pulse 99, full; temp. 101·3°. Takes a great deal of milk, nothing else. Skin painted with arg. nit.

"24*th.*—Very restless and delirious all night. Face greatly swollen on both sides, also his neck. Wound is quite dry; small slough is forming at inner angle of left eye. Pulse 160 and very weak; temp. not taken. Towards the afternoon the erysipelas subsided a good deal, but patient continued delirious. He was very suspicious and excited and obstinate. He would sit up in bed resting his head, which was quite straight and quiet, on his right hand and talk loudly. Once he declared that his medical attendants were trying to keep his case to themselves, but they would not be able to do it. Everybody would hear of it. There were no symptoms of arachnitis.

"25*th.*—This morning the swelling is again present, together with some dusky redness. The right upper eyelid contains a good deal of pus. When the patient moves there is a great deal of spasm and twitching of the muscles. Patient very apathetic. Pulse weak and not rapid.

"26*th.*—About the same; unable to swallow; fed with enemas. Bowels opened unconsciously; no convulsions. Patient can now move his arms but slightly and jerkingly, and grasps feebly. Lies quiet. Pulse very weak and irregular; temp. not taken. Tongue very dry, hard, brown and cracked."

This is the last note made by Mr. Mears, who paid great attention to the case throughout. Death took place a few hours later. The post-mortem examination was conducted by Dr. Sutton. The visceral appearances were those due to congestion from the erysipelas, and did not present anything to account for the result of the case.

The brain and spinal cord were carefully examined by Dr. Jackson, Dr. Sutton, and Mr. Rivington, and were reserved by

Dr. Sutton for microscopic examination. Unfortunately the specimen seems to have been mislaid, for no note could ever be discovered concerning it. From the firmness of the spinal cord in the cervical region, both in its entirety and when cut into slices, Mr. Rivington suggested that it was sclerosed, and Dr. Sutton thought this very probable. The ends of the spinal accessory nerve were found separated, and had been completely divided.

Remarks by Mr. Rivington.—My reasons for following Mr. De Morgan's mode of operation were that it afforded more room to the operator than seeking for the nerve in front of the sterno-mastoid, and seemed less likely to implicate the jugular vein. I performed both operations previously on the cadaver, and found that I could easily reach the spinal accessory and divide it in front of the sterno-mastoid, but that I should be obliged to work in a hole, and if any troublesome bleeding occurred from the sterno-mastoid branch of the occipital artery, it would probably be necessary to enlarge the incision and divide the external jugular vein, &c., before the artery could be comfortably tied or twisted. By Mr. De Morgan's method no important nerve or vessel need be touched, and it was quite easy to trace the spinal accessory and isolate sufficiently for the purpose. The termination of the case was disappointing. The operation was not performed under the carbolic spray, as it did not seem to be necessary, and would have tended to obscure the dissection. There had been a few scattered cases of erysipelas in the hospital, but none in the part of the building in which the patient was located. By what channel he became affected I could not determine. In reference to this matter it has occurred to me to put the question whether, assuming the cervical cord to have been the subject of sclerosis, a predisposition to vascular disturbance from slight irritation had not been created. Seeing that the spinal accessory nerve, and indeed the other cervical nerves, had become the channel for the transmission of irregular impulses due to the central disease, and set going perhaps by any slight peripheral impressions, might not a *small* amount of irritation in the wound, and a *small* quantity of peccant matter in the blood, cause abnormal disturbance to the vaso-motor centre and vaso-motor paths in the cervical portion of the spinal cord? The erysipelas spread no further than the head and neck, and shoulder and upper part of the thorax—over the regions covered by the nerve-supply from the cervical plexus and cranial nerves arising from the medulla, and inasmuch as the reflex connection between sensory and vaso-motor nerves is very intimate, it appears to me that a good deal of the vascular disturbance may be accounted for in this manner. Admitting that some

materies morbi had entered the blood through the wound, I cannot help thinking that but for the neurosis the patient would have recovered, for many cases worse than his get perfectly well, and deaths from erysipelas have not been by any means frequent in my experience at the hospital. As the spasms noticed after the operation completely subsided in a few days, and did not recur, it is fair to assume that he would have been relieved of his complaint if he had recovered from the erysipelas."

Remarks by Dr. Hughlings-Jackson.—"It is worthy of note that the erysipelas did not set in till nearly a fortnight after the operation, when the wound had nearly healed. The patient shivered a good deal the morning after the operation, and the temperature stood at 103°·2. Does the interval between the shivering and the appearance of the erysipelas represent a stage of incubation?"

Abstracts of British and Foreign Journals.

Kirchoff on Cerebral Glosso-pharyngo-labial Paralysis. (*Archiv f. Psych.* Bd. xi. p. 132.)—A man, aged 24, while perspiring profusely, stepped into water for a bathe. Scarcely had he done so, when he became giddy, had convulsive tremors in his extremities, lost the power of speech, and was unable to swallow. Saliva flowed from his mouth, and his face was drawn to the left. These symptoms disappeared in a few days, but a week later he had a similar attack. On admission into hospital, July 29, 1877, it was observed that he articulated with difficulty; labials and gutturals especially were troublesome; linguals he spoke with comparative ease. The lips were moved little in speaking; he could not whistle, but was able to approximate the lips. Saliva flowed from the mouth, and there was excessive secretion of tears. The tongue was not protrusible more than 1 centimetre from the mouth, and it was moved clumsily in the act of biting. At the time of examination swallowing was unimpeded, but the glottis was closed tardily. Patient often laughed without occasion. There was disease of the mitral valve.

During the progress of the case there were marked remissions and exacerbations. On November 2 swallowing was only possible with the head bent back, and even then was difficult. Saliva flowed from the mouth continually, and in his laugh, crowing and whistling sounds were often heard. His arms were shaky. On December 25 his face was suddenly drawn to the right, and his left arm and leg became powerless. Chronic convulsions occurred from time to time up to his death, on Jan. 5.

The post-mortem revealed embolic softening of the posterior two-thirds of the right corpus striatum (caudate nucleus ?), the underlying internal capsule, the outer segment of the lenticular nucleus, the claustrum, external-capsule, and island of Reil. The focus of softening in the lenticular nucleus was distinguished from the other softened portions by being surrounded by a wall of compact sclerosed tissue. Very careful microscopic examination failed

to show any disease of the medulla or pons. The author attributes the glosso-labial paralysis to the lesion of the lenticular nucleus; and the hemiplegia to the quite recent lesion of the caudate nucleus, internal capsula and other parts. Cases of bilateral affection of the face, tongue, and throat, caused by unilateral lesion of the cerebrum, are rare; the author cites two, recorded by Lépine and Magnus respectively.

Virchow on Progressive Facial Hemi-atrophy.—Virchow (*Berliner klin. Wochenschr.* 1880, No. 29, and abstract by Bernhardt in *Centralbl. f. med. Wissensch.* 1881, No. 3) reports two cases of this disease under the name of "neurotic atrophy." The first was a man, aged 42, whose face became atrophied on the left side in his ninth year. Virchow had seen the same patient twenty-one years previously, during which time the disease had made no appreciable progress. The second case was a woman, aged 41, in whom the disease had shown itself sixteen years ago, after an inflammatory affection of the skin of the left eye. Shortly before this the patient had fallen on the back of the head, and during childhood had often suffered from furunculus. There was atrophy not only of the left side of the face, but of a large area extending from the middle line of the back (between the fourth and seventh dorsal vertebræ) outwards over the left infra-spinous fossa to the shoulder, and thence down the back of the arm to the ulnar side of the forearm. The area of distribution of the left radial nerve was markedly atrophied. Tactile sensibility was normal; at one time sharp twinges of pain were felt in the eye and arm, but at the time of examination these had given place to a numb feeling in the arm and a feeling of cold in the little finger. The author regards the disease, in its origin at least, as a peripheral affection of the nerves. The morbid process, inflammatory or otherwise, may spread upwards till it reaches the ganglia in the basis cerebri or spinal column. The atrophic areas correspond to the course of various nerves, but not to the whole area of distribution of these nerves; certain fibres only of the nerves become diseased. The author remarks that there are few things in nerve pathology more striking than the unequal affection of the fibres of the same nerve in this disease. He regards lesion of the trigeminus as the starting point of the malady. The bones atrophy if the affection commences in the earlier years of life.

Flascher (*Berliner klin. Wochenschr.* 1880, No. 31) describes a case of *bilateral* facial atrophy, occurring in a woman aged 23.

When an infant she fell and injured her forehead, and shortly afterwards had an attack of measles, without eruption. It was soon after this that the facial atrophy appeared. The trigeminus, as a whole, was not implicated, but only particular branches of it, and only particular fibres of these branches. In the atrophic places the vaso-motor system was unimpaired, tactile sensibility diminished, the secretion of sweat paralysed. The bones were atrophied, so also the facial muscles and the masseters and temporals. There was external strabismus of the left eye, the pupil of which was irregular, widely dilated, and non-reactile; sight was defective, and the optic disc atrophied and white. The weakness of sight and strabismus were detected about the same time as the facial atrophy. Flascher, too, regards the disease as a peripheral affection of the nerves, coming on in this case as a sequela of measles.

Hammond (*Journ. Nerv. and Ment. Disease*, April, 1880) observed a case of progressive facial atrophy in a girl, aged 14. The affection was of gradual growth, and did not attract attention till she was 12. There was a decided difference in the size of the two sides of the face. On the left side there were three depressions, two near the angle of the mouth, and one above and slightly in front of the ear. The left half of the tongue was much smaller than the right, and the palatine arch was flatter on the left than on the right side. Muscles supplied by the motor branch of the fifth nerve, by the facial and by the ninth, were atrophied; microscopic examination of them showed atrophy without degeneration. There was no appearance of paralysis. The skin, hairbulbs, cellular tissue, and even the bone (temporal) were also atrophied. There were occasional paroxysms of numbness lasting a few minutes in the left side of the face. Defective sensibility was found only in a small region over the left half of the orbicularis oris. The author considers the nuclei of the trigeminus, facial, and hypoglossal as the primary seat of the disease, which consists essentially in lesion of the trophic cells of these nuclei.

Eulenburg on Facial Spasm. (*Centralbl. f. Nervenheilk.* 1880, No. 7, and *Cbl. f. med. Wissensch.* 1880, No. 47.)—A woman, aged 25, after an attack of transient aphasia, without loss of consciousness, became subject to violent and painful contractions of the muscles of the left face, especially of the orbicularis palpebrarum. In severe attacks the convulsions spread to the muscles supplied by the left spinal accessory, and to the flexors of the left hand and

digits, and on rare occasions to the right side of the face and neck. There was hyperalgesia of the left face, and numerous painful points were found along the course of the trigeminal and facial nerves. Galvanisation, bromide of potassium, injections of atropine, morphine and curare, and neurotomy of the supra-orbital nerve having proved ineffectual, the facial nerve was stretched. The operation cured the spasm, but left almost total paralysis of the facial nerve. The muscles of the underlip were only partially paralysed, and those supplied by the posterior auricular nerve were quite intact. A curious result was the abolition of the sense of taste in the anterior half of the left side of the tongue; sensibility and salivary secretion were normal. After a time spontaneous, intermittent fibrillary contractions, tonic-clonic in character, appeared in the depression of the lower lip, which soon became the seat of a contracture. Fifteen weeks after the operation, the sense of taste returned. As lesion of the chorda tympani is out of the question in this case, Eulenburg attributes the loss of taste to lesion of a filament, which, according to anatomists, is sometimes found connecting the peripheral branches of the glossopharyngeal and facial nerves.

Mossdorf on Aphthongia.—This was the name given by Fleury in 1865 to a variety of aphasia, characterised by spasm of the muscles supplied by the hypoglossal nerve whenever the patient attempts to speak. In Mossdorf's case (*Cbl. f. Nervenheilk.* 1880, No. 1, and *Arch. de Neurologie*, 1880, No. 2), on the patient, a boy aged 17, attempting to speak, the hyoid and abdominal muscles became cramped, respiration ceased, and through the half-opened mouth the tongue was seen contracted, its tip firmly pressed against the lower incisors, and its dorsum against the palate. In a few moments the abdominal muscles relaxed, and respiration was resumed, but the spasm of the tongue and hyoid muscles persisted. The movements of the tongue, lips, and face were, with this exception, quite normal. Latterly the boy was unable to reply to his parents, or even read aloud when alone. The difficulty of speech showed itself when he was six years old, and was attributed to fright. A cure was effected by galvanisation; the negative pole was placed in the nape of the neck, and the positive pole slowly moved up and down the spine.

Schultze on Multiple Cerebro-Spinal Sclerosis and General Paralysis.—Schultze (*Arch. f. Psych.* Bd. xi. p. 216) relates a case

which, during the first two years of its course, was looked upon as cerebro-spinal sclerosis. The symptoms were as follows: Tremors of the extremities during voluntary movements, paresis of the legs and of the right arm, pains in the extremities, impaired sensibility of the right arm, at times incontinence of urine, hallucinations of sight, diplopia, vertiginous seizures, epileptiform attacks, slow, stuttering, monotonous speech. There were marked remissions in the symptoms, and twice the gait of the patient, who, we may mention, had suffered from syphilis, so far improved that he was discharged. On his third admission into hospital, mental disturbances for the first time showed themselves. He became acutely maniacal, had grandiose ideas, and presented up to the time of his death the typical symptoms of general paralysis. It is a remarkable fact that tremors were never observed during voluntary movements after the appearance of the mental disorder. On *post-mortem* examination there was found, in addition to the usual accompaniments of general paralysis (diffuse hyperplasia of the connective tissue and atrophy of the cerebrum), multiple sclerosis of the cord and diffuse sclerosis of the brain. In another case of general paralysis, the author found small patches of sclerosis in the cord, and diffuse sclerosis in both cord and cerebrum.

The concurrence of the two diseases, multiple sclerosis and general paralysis, rare though it seems to be (a case by Claus is reported in 'BRAIN,' Vol. II. p. 142), is only what might be expected from their pathology, for they are both characterised by a widespread hypertrophy of the neuroglia with the formation of granule-cells. Sclerotic changes in the cord, particularly in Gall's columns, and the pyramidal strands occur frequently in general paralysis.

The first case offers a few subsidiary points of interest:—(1.) the cord in the fresh state appeared quite normal; it was only after immersion in Müller's fluid that the sclerosed patches became visible. (2.) Though the whole area of the posterior columns was sclerosed in the lower part of the dorsal region, and again in the lower part of the cervical enlargement, there was but slight secondary degeneration of Goll's columns. The lateral pyramidal strands were degenerated through the greater part of the cord, but in the upper cervical region were quite intact, which shows that the degeneration was of spinal origin. The relation of multiple sclerosis to secondary degenerations is still far from clear. Schultze suggests that the pyramidal strands and Goll's columns are, from their anatomical constitution, the parts that suffer most severely in general hyperplasia of the connective tissue of the

cord. (3.) Corresponding to the isolated atrophy of the interossei muscles of the right hand, there was lesion of the right anterior cornu in the lower part of the cervical enlargement, which coincides with other observations bearing on the localisation of the spinal centres for the small muscles of the hand.

Seeligmüller and Schmid on Hereditary Ataxia.—Seeligmüller relates the histories of two brothers who suffered from this disease. The younger, aged 26, showed symptoms of ataxia of the lower extremities when 12 years old. Ten years later, nystagmus developed, and quite recently slight ataxia of the superior extremities had been observed. The ataxia of the legs was visible both in walking and in standing with the feet together (static ataxia). The movements of the eyes had not the usual rythmical character, but were more irregular and jerking, and corresponded with Friedreich's atactic nystagmus. There were no fibrilly contractions, except slight ones in the protruded tongue; sensibility and electrical irritability of nerve and muscle were normal; there were no paralyses, contractures, or atrophies. The secretion of saliva was abnormally abundant. This case differs from those described by Friedreich in the following points: (1) There was no ataxia of speech; (2) the patellar reflexes were heightened, not absent; (3) there were cerebral symptoms, in the shape of psychical manifestations, *e.g.* forgetfulness, tendency to reverie, perverted sexual instincts, and melancholia when 16 years old; and (4) there was occasional impairment of the vesical and anal sphincters. In the elder brother, aged 28, the nystagmus and the static and locomotor ataxia of the lower extremities were less marked. The upper extremities were unaffected. The patellar reflex was lively, sensibility intact. The patient was forgetful and absent in mind; he spoke very rapidly, but sometimes not very intelligibly, as he would hurry from sentence to sentence without finishing them. Father and mother were cousins, and nervous diseases were common in the family.

Schmid's case (*Centralblatt f. med. Wissensch.* 1880, No. 25) was a boy of 18, whose gait five years before had become uncertain and peculiarly rapid, and at length resembled that of a drunken man. There was marked locomotor and static ataxia of the lower extremities, and slight incoordination in the movement of the arms. Closing the eyes, or being in darkness, did not intensify these symptoms. Muscular power and electrical irritability were normal, the superficial reflexes unaffected, the patellar reflex

absent. Speech was halting and inarticulate, and the eyes exhibited nystagmic movements. Sensibility was intact, and the sphincters acted naturally. There were no mental symptoms. At a later period the patient became unable to walk, and slight sensory disturbances were noticed in the lower extremities. A younger brother, when 11 years old, began to show symptoms of locomotor ataxia, and his patellar reflex disappeared. A sister, aged 9, is healthy up to the present. There was no special tendency to neuroses in the family.

Remak on Localised Ataxia and Ephidrosis. (*Berliner klin. Wochenschr.* 1880, No. 22, and *Centralbl. f. med. Wissensch.* 1880, No. 51.)—A man aged 38, infected with syphilis twelve years previously, had suffered for five years from gradually increasing anæsthesia of the right forearm, hand, and fingers. The movements of the limb were in a high degree ataxic, but not diminished in strength. Shortly after the appearance of the ataxia, excessive secretion of sweat was observed on the right half of the head, and slight reddening of the face. There was myosis of the left eye. Lately symptoms had developed which suggested commencing tabes—unsteadiness of gait in darkness, or when eyes were shut, a feeling of numbness in right sole, difficulty in micturition, absence of patellar reflex. The author diagnosed a patch of sclerosis in the right posterior half of the cervical enlargement, with secondary changes in both posterior columns. The unilateral ephidrosis may be due to lesion of the cerebro-spinal secretory centres, or perhaps to lesion of the cervical sympathetic.

W. J. Dodds, M.D., B.Sc.

Gaucher on the Morbid Anatomy of Diphtheritic Paralysis.—Gaucher (*Journal de l'Anatomie*, No. 1, 1881) reports the result of an investigation of two cases of Diphtheritic Paralysis. In one of these no lesion was discoverable, either in the muscular or nervous system. In the second, however, there were well-marked appearances corresponding in most points with those described by Déjerine as characteristic of this form of paralysis. Déjerine had found that the essential lesion was a parenchymatous neuritis of the anterior roots of the spinal cord, characterised by a segmentation of the myeline and multiplication of the nuclei in the sheath of Schwann with complete or partial disappearance of the axis cylinder. Further, in the anterior horns, the cells were less numerous, rounded and deprived of their processes.

Gaucher concludes from his own researches that there are never lesions of the muscular system, but that the lesions are confined to the nervous system, which, however, are not constant.

Apart from general congestion of the nervous centres which is not characteristic, these lesions are specially marked in the anterior roots, but generally only a certain proportion of the nerve tubes are implicated. The lesions are total disappearance of the myeline and considerable proliferation and enlargement of the nuclei of the sheath of Schwann.

The axis cylinder, however, is continuous throughout, distinct and without alteration. The neurilemma is free from lesion.

Morgan and Dreschfeld on Idiopathic Lateral Sclerosis.—In the *British Med. Journal*, January 29, 1881, Dr. Morgan relates the clinical history of three cases of so-called "spasmodic spinal paralysis" (Erb), or "tabes dorsal spasmodique" (Charcot). In one of the cases which died, an examination of the spinal cord was made by Dreschfeld, who found the following morbid appearances. The cord examined in the fresh condition, ten hours after death, presented no abnormality, except softening in the lower dorsal region. After hardening in bichromate of ammonia, sections of the cord showed light-coloured patches in each lateral column, specially marked in the dorsal region and less so in the cervical and lumbar regions. After staining with carmine the sclerosed patches were seen in the cervical region to be in close relation internally with the grey matter; anteriorly they did not extend up to the anterior horns, and externally did not reach the surface of the white substance. The grey matter of the anterior and posterior horns was normal. The patches in the dorsal region were more distinct and extended further anteriorly. In the lumbar region the sclerosed patches were situated close to the outer side of the posterior horns, extending almost up to the outer border of the lateral columns posteriorly, but only a little way in an anterior direction. The grey matter here, as in the cervical and dorsal regions, was normal.

Some of the microscopic sections were submitted to M. Charcot, who characterises this case as the first in which sclerosis has been demonstrated in the lateral columns without implication of the grey matter or posterior columns.

Dalton on the Visual Centres.—Dr. J. C. Dalton (*The Medical Record*, March 26, 1881) gives the details of two experiments on

dogs, with reference to the localisation of a visual centre in the cortex.

In the first experiment the cortex was destroyed at a point corresponding with the bend of the second external convolution, and, as was found post mortem, extending also into the two convolutions situated between this and the fissure of Sylvius. As the result of this lesion there was complete blindness of the opposite eye, with apparently normal vision in the eye of the same side. Motor power and general sensibility were unaltered. The animal died on the third day from encephalitis.

In the second experiment the lesion was more accurately confined to the second external or angular convolution, extending only partly into the convolution above and behind it. As the result of this lesion there was blindness of the opposite eye, and this only. The animal lived for twelve days, continuing blind in the eye opposite the lesion to the end.

The only particulars in which these results differ from those of Ferrier on monkeys, is their persistence, in regard to which he makes some suggestions and criticisms.

But the evidence he says warrants the following conclusions:—

1. Extirpation of the angular convolution causes loss of visual perception on the opposite side.

2. This operation is not followed by any disturbance of the intelligence, attitude, power of locomotion or general sensibility.

3. It does not interfere with the local sensibility of the retina or conjunctiva, the reaction of the pupil to light, nor with the normal consentaneous movements of winking. Its effects are, therefore, confined to the exercise of visual sensibility.

Christiani on the Influence of the Brain on Respiration, &c.—Christiani (*Acad. d. Wissensch. zu Berlin*, February, 1881, Sep. Abdruck) describes certain experiments relating to the influence of the brain and nerves on the respiratory mechanism; and also the effects of certain lesions on the animal's powers generally.

He finds that the inspiratory mechanism is stimulated by irritation, electrical, mechanical and otherwise, of certain sensory nerves, the optic, auditory, tactile, as well as by means of certain vagus fibres. Irritation also of a point in the wall of the third ventricle close to the corpora quadrigemina causes deep and quickened inspiratory movements. On the other hand irritation of the trigeminus, or of a point in the anterior tubercles of the corpora quadrigemina,

just under the aqueduct of Sylvius, causes expiratory movements. This action is associated with contraction of the pupil, whereas the inspiratory centre is associated with dilatation.

These "centres" are modified in their action by certain drugs, increased by strychnine, depressed by chloral.

Inspiratory action is also caused by irritation just posterior to the anterior tubercles of the corpora quadrigemina, confirmatory of previous researches on this subject.

Rabbits, after removal of the hemispheres and corpora striata, remain capable of locomotion, apparently even spontaneously. The action to sensory stimulation seems increased.

When section was made just anterior to the corpora quadrigemina, or injury made in the region of his "inspiratory centre" co-ordinated locomotion was no longer possible.

After the removal of the optic thalami and anterior surface of the corpora quadrigemina, reflex movements from mechanical stimulation of the hinder extremities were difficult to obtain, but irritation of the trigeminus or pinching the tail caused the utterance of peculiar cries. After section posterior to the corpora quadrigemina tetanic spasms occurred, and were readily excited by the slightest touch as in strychnine poisoning. This tetanic condition subsides, and leaves increased reflex excitability.

Animals possessing only the medulla, cerebellum and pons, make defensive movements if the nose or conjunctiva is irritated.

After destruction of the third ventricle or section posterior to the corpora quadrigemina, strychnine appears to act less energetically. Strychnine seems specially to act on the complex ganglion centres of the optic thalami.

Adamkiewicz on the Influence of Sinapisms on Sensibility.—Adamkiewicz (*Berlin. klin. Wochensch.* No. 12, 1881), in a lecture to the physicians of Cracow, relates some interesting observations on the influence of mustard plasters on anæsthesia and normal sensation. In previous researches he had shown that the secretion of sweat was dependent on a bilaterally functioning centre in the medulla oblongata, acting both through cerebro-spinal and sympathetic tracts. Taking up the subject of "transfer" of sensibility, so well known in connection with metallo-therapy, he found that sinapisms produced precisely the same effect as metals.

In a case of organic hemianæsthesia (proved by a post-mortem examination to be dependent on hæmorrhage into the lenticular nucleus and internal capsule), he succeeded by sinapisms applied to

the anæsthetic side, in restoring sensibility on the whole of the paralysed side, lasting several days. The restoration was not permanent. There was no transference.

In the various forms of hysterical anæsthesia the results were variable. In certain irregularly distributed anæsthesiæ, sinapisms restored sensibility for a greater or less duration. This is not in relation with the condition as to vascularity.

In a case of hysterical hemianæsthesia, sinapisms produced all the phenomena of transfer. After a few days the old condition returned, but on renewal of the sinapisms sensibility was completely restored without being lost on the other side.

In another case of hemianæsthesia and hemiparesis complete transfer occurred also with lowering of the temperature 5° on the sound side, and corresponding rise on the side formerly anæsthetic.

Following out indications supplied by these observations he investigated the influence of sinapisms in normal individuals and found that the sensibility for touch and pain increases on the side of irritation, and diminishes correspondingly on the symmetrically placed point on the other side. Hence it appears these forms of sensibility are bilaterally organised in the nerve centres, and functionally antagonistic, as shown by the diminution of the one when the other is increased. The result also seems to be that the sense of touch and pain is independent of the richness of the part in nerves, and is a purely qualitative function of the skin.

As regards the thermal sense, however, it was found that though it is also rendered finer by sinapisms at the point of irritation, yet it is not correspondingly diminished on the other side. The thermal sense, therefore, does not appear to be bilaterally organised.

In a short appendix to the paper, data are given showing that the diminution of sensibility on the side not stimulated by sinapisms is not related to the circulation, though irritation of the skin causes bilateral changes in the circulation just as it does in the case of sweat-secretion.

Grainger Stewart on Peripheral Paralysis of Hands and Feet.—Dr. Grainger Stewart (*Edin. Med. Journ.* April, 1881), describes three cases of the co-existence of sensory, motor and trophic disorders in the hands and feet, most marked in the distal points, and not corresponding to the areas of distribution of particular nerves.

In one of the cases, fatal from pneumonia, degeneration was found in the cervical and slightly in the lumbar enlargement,

affecting the columns of Goll and the direct cerebellar tracts of Flechsig. The grey matter was unaffected. The median, ulnar and tibial nerves also exhibited morbid appearances microscopically, the process consisting in a breaking up of the axis cylinder. The morbid process did not affect the large cords of the plexuses.

Dr. Stewart believes the degeneration commenced in the periphery of the nerves and spread upwards.

He gives references to similar cases recorded in various quarters, and indicates the points of difference between this affection and other forms of paralysis. Recovery is the rule, but it may in some cases lead to atrophic paralysis, or extend into the spinal cord and cause death by affecting vital centres.

James on Deafmutism and Dizziness.—Professor James (*Harvard University Bulletin*, April 1, 1881) publishes an interesting preliminary note of an investigation he has made as to the sense of dizziness in deaf mutes. He was led to the inquiry by the known relationship between the semicircular canals and the sense of rotation, and by the speculation that, therefore, deaf mutes would be deficient in this respect. The result of actual experiment was to confirm this in the most marked manner. A very large number of deaf mutes are either wholly incapable of being made dizzy by the most violent rotations, or experience but a slight and transient giddiness. Others are strongly and normally affected. The deficiency seems quite independent of the age at which the deafness begins, semi-mutes and congenitals being found indifferently in all classes. Disorders of locomotion are also common among deaf mutes, a subject which deserves careful attention from physiologists.

From his researches Dr. James expresses the opinion that the normal guiding sensation in locomotion is that from the semicircular canals. This is co-ordinated in the cerebellum with the appropriate muscles, and the mechanism becomes structurally organised in the first few years of life. If the guiding sensation is abolished by disease, compensation must be effected through sight and touch. This, however, is affected but slowly, and hence for many years the patient's gait is uncertain, especially in the dark. When the defect is congenital, the cerebellar mechanism is organised from the outset in co-ordination with tactile sensations, and no difficulty occurs.

An extended examination of typical cases is desirable, in order to test the value of this hypothesis.

Brachial Monoplegia (Traumatic).—Stimson (*Archives of Medicine*, April 1881) records an interesting case in which trephining was practised for a cerebral abscess resulting from traumatic lesion of the right side of the head above the ear, two months and a half previously. Besides the general indications of cerebral abscess, there were localising symptoms in the form of wrist-drop on the left side.

The Rolandic line was found to pass about half an inch in front of the upper end of the scar. A small perforation of the skull was found at this point, and on the trephine being applied just anterior to the opening, an abscess was found a quarter of an inch below the surface. About two ounces of pus were withdrawn after incision into the dura and brain substance. The patient died nine hours after the operation.

The seat of the abscess was at the point immediately posterior to the lower third of the ascending parietal convolution—the centres for the hand and wrist.

DAVID FERRIER.

Buzzard on the affection of Bones and Joints in Locomotor Ataxy, and its association with gastric crises. (*Transactions of the Pathological Society of London for* 1880; *British Medical Journal*, March 5th, 1881.)—Since 1868, when Professor Charcot first directed attention to the peculiar arthropathy associated with locomotor ataxy, the subject has engaged the attention of numerous observers abroad, especially in France. It is remarkable that in England there have been but few references to it. With the exception of a brief record of a case by Dr. Clifford Allbutt (*St. George's Hospital Reports*, 1869), Dr. Buzzard was the first to refer to it, in a clinical lecture upon a typical case of the kind, published in the *Lancet*, August 22, 1874. Since that time he has collected seven more cases, notes of which are published in the communications before us. The following is a brief description of these:—

1. A man with complete disorganisation of the right hip-joint, the head and neck of the femur having entirely disappeared in a few months. In this case a peculiar splint of bone had developed in the quadriceps femoris muscle.

2. A woman with spontaneous fracture of both femora.

3. A woman with disorganisation of both knee-joints, and incipient arthropathy of the right shoulder-joint.

4. A man with complete disorganisation of the left knee-joint.

5. A man with disorganisation of the left knee-joint.

6. A man in whom the head of the left humerus had disappeared.

7. A woman with arthropathy of the left hip-joint. The head of the femur had disappeared.

8. A man with arthropathy of the right knee-joint.

This is a simple list of the cases. Reference must be made to the papers for ample details, which it would occupy too much space to reproduce here. It is sufficient to say that the descriptions leave no doubt that they were all typical cases of tabes dorsalis.

A very interesting and original suggestion is made by Dr. Buzzard in reference to these cases. He was much struck by the fact that two of the female patients referred to above, who were under his care at the same time, suffered from the typical *crises gastriques* of Charcot. His experience told him that both the arthropathy and the gastric crises were very exceptional symptoms in tabes dorsalis. Their coincidence in two cases suggested to him the idea of inquiring whether there were grounds for supposing the association to be something more than a strange coincidence. The result of the inquiry is very remarkable. He has found that out of thirty cases of affections of bones and joints in tabes (which includes eight of his own) no fewer than fourteen, or nearly half, were affected with typical gastric crises. Whilst searching for data he came across a remark of Professor Ball in his paper on the subject (*Gazette des Hôpitaux*, 1868–69), in which he notes that in one-fourth of his cases "visceral troubles" were associated with the articular lesions.

Buzzard shows that the idea of Charcot that the articular lesions would be found to depend on lesion of the ganglionic cells in the anterior cornua, has not been supported by investigation, and he adduces numerous reasons which appear to prove conclusively that it must be abandoned. Starting with the suggestion that the gastric crises may be reasonably supposed to depend upon lesion of the roots of the vagi homologous with the lesion of the posterior columns of the cord, Buzzard suggests that the lesion of joints and bones probably depends upon invasion of a part of the medulla oblongata close to the roots of the vagi. He asks, Is there something which we may call provisionally a trophic centre for the osseous and articulatory system in the immediate neighbourhood of the roots of the vagi? The discovery of such a centre would materially help us, he adds, to explain the remarkable association of cardiac complications with the joint affection of acute rheumatism, as well as the sweating characteristic of this disease, and the

occasional hyper-pyrexia which occurs in it, and it might also help us to throw light upon the pathology of arthritis deformans.

It is interesting to note that after Dr. Buzzard had called attention at the Pathological Society to the frequency of association of gastric crises with the arthropathy of tabes dorsalis, the President (Mr. Hutchinson) related some particulars of a case which also showed this association; and Dr. Sturge referred to another, in which a similar association was to be found. When, later on, Buzzard was pursuing the subject, he met with four more cases of arthropathy, in three of which again typical gastric crises formed part of the history.

In a work by Dr. X. Arnozan, recently published, 'Des Lésions trophiques consécutives aux maladies du système nerveux,' exception is taken to Buzzard's hypothesis on the grounds that gastric crises are so common that the association is not important, and that there is no evidence that they are caused by sclerosis of the roots of the vagus. Arnozan thinks the seat of osseous lesion must be sought in the sensory regions of the cord.

Buzzard on Acute anterior Polio-myelitis in infants and adults. (*Lancet*, December 4 and 25, 1880; *Medical Press and Circular*, January 26, February 2, 9, 16, 23, March 2, 1881.)—In the course of two lectures, delivered at the National Hospital for the Paralysed and Epileptic, several patients were exhibited, and the symptoms, diagnosis, prognosis, etiology, and treatment of the disease were discussed. In reference to diagnosis, Buzzard points out that muscles in the district of a particular nerve are often found very unequally affected, some being completely paralysed, others but slightly affected. This, with the absence of cutaneous anæsthesia, points to a central origin, not an affection of a mixed spinal nerve-trunk. Where the quadriceps extensor femoris is paralysed there is absence of patellar tendon reflex. Buzzard has frequently observed that during regression many muscles recover their excitability to voluntary impulses long before they respond to the induced current, and he makes the following original suggestion as a possible explanation of this anomaly. He points out that according to Huguenin the anterior root is composed of fibres of which, although the greater part are connected with the ganglionic cells in the anterior horn, some ascend in the lateral column distinct from such connection. These fibres not having their trophic centre in the ganglionic cells, but higher up, would escape, he suggests, all but a temporary obstruction of function,

and would convey motor impulses to the muscles sufficient to cause movement. He lays stress on the condition of patellar tendon reflex as a convenient mode of diagnosis between this disease and cerebral paralysis. In the former it is absent, in the latter either unaffected or still more often in excess. Two patients were shown to illustrate this point: a hemiplegic form of infantile paralysis, and a case of cerebral hemiplegia in an infant.

Buzzard demonstrated the reaction of degeneration, and suggests that simply by using the letter Z (German Zückung) the English notation can be assimilated to the German, and comparison much facilitated. He uses K—kathode; A—anode; S (shutting—schlieszung); O (opening—oeffnung). He remarks that the Germans have set us an example by adopting Faraday's terms, Kathode and Anode.

He relates (in reference to diagnosis) the case of an infant who suddenly lost the use of a lower extremity after a little febrile disturbance. A large amount of uric acid was found in the urine, accounted for, he thinks, by the child having been nourished on a milk food containing an enormous quantity of sugar. Buzzard supposes that minute crystals of uric acid, or some equivalent, had got deposited in the loose connective tissue between the muscles (expansions of the lymphatic system), and set up some inflammation. The suggestion of this as a cause of symptoms he owed to Dr. Burdon-Sanderson.

A patient was shown with paralysis of the right arm, left leg, and *right side of the face*. The latter unusual complication of this disease he attributes to extension of the affection upwards to the nucleus of the portio dura, and suggests that the reason why we do not often witness the association is because the disease when it invades the bulb is likely to prove fatal by involving the roots of the vagus also. Cases of unexplained death may more often occur from this cause than is commonly supposed. Buzzard makes the important observation that such a thing as monoplegia is extremely rare in this disease. Limbs supposed to be unaffected are only comparatively so, as shown by lowered faradic excitability, and absence of tendon reflex.

He notes that alcohol must be absolved from the charge of causation. The patients most numerously affected are children. Some adult cases brought forward by him were abstainers.

The atrophy of the muscles is, for a long while at least, simple atrophy. It is the affection of the ganglionic cells, and nerve fibres proceeding from them, which is the serious lesion; and

Buzzard does not see much use in stimulating the *muscular* fibres by electrical currents. Indeed there is a disadvantage, at least in the early stage of the disease, for the stimulation of the skin being conveyed to the cord tends to irritate the ganglionic cells in the anterior horn.

Nor, when deformities are commencing, would he encourage voluntary movements of a limb, for by so doing overaction of the muscle least affected is encouraged, and the deformity is thereby increased. He enjoins rest in bed, ergot as early as possible, a mild constant current (stabile) to the spinal cord, and a light apparatus to check deformity as far as possible.

Buzzard on Tendon Reflex in the Diagnosis of Diseases of the Spinal Cord. (*Lancet*, November 27th and December 4th, 1880.)—In previous papers (*Lancet*, July 27th and August 4, 1878; *Brain*, July, 1878; *Lancet*, January 18, 1879, January 10, February 14, March 20, and April, 1880) Buzzard has described in detail the knee and foot phenomena of Westphal and Erb. The present paper is a contribution to the arguments which the clinical facts afford in favour of the reflex mechanism of the phenomena. Recognising that some eminent physiologists discard this view, principally on the ground that the interval of time between the blow and contraction is too short for a true reflex, Buzzard thinks that either there may be some source of fallacy in measurement, or that a shorter period of time may be required for conduction through the centre where, as in the present case, the centripetal stimulation is strong and rough. The absence of tendon reflex in tabes, along with preserved skin reflex, shows, he suggests, that the path of conduction must differ in the two phenomena, and that they cannot properly, therefore, be compared as regards time.

Buzzard points out that a convenient mode of testing the patellar tendon reflex is by lightly touching the patient's thigh with the left hand, whilst with the right a blow is given with a percussion stethoscope or hammer on the ligamentum patellæ, the patient being seated with his foot resting firmly upon the ground, and slightly advanced. The quadriceps muscle is felt to contract. The mode is especially convenient for the examination of females.

By a rough diagram Buzzard shows the nervous arc of communication extending from the tendon to the posterior roots, anterior horn, anterior roots, and thus to the muscle. He remarks that the integrity of this arc ensures the contraction of the muscle (if *it* be

healthy) to a blow on the tendon. On the other hand, the response to a blow *nearly always* proves that the arc is not seriously interrupted; but he makes the important observation that there is an exception to this rule. There may be atrophy of some ganglion cells in the anterior horn. If this lesion existed alone, tendon reflex (as shown by another diagram) would be abolished; but if the lateral column be sclerosed, the inhibitory influence from above is so much interrupted that reflex occurs. In cases, therefore, of lateral sclerosis with muscular atrophy, and also in cases of tabes dorsalis with lateral sclerosis, we may get the patellar tendon reflex preserved although the nervous arc is not intact. He ascribes the exaggeration of tendon reflex to the interception of controlling influence which comes down through the lateral column, and points out that the absence of this influence (e.g. in a case of hysterical paralysis) produces similar effects to its mechanical interruption by means of sclerosis, though in the former case they are only temporary. He points out that in spastic paraplegia, secondary to myelitis, the exaggeration of reflex may be connected with a temporary interruption (something short of sclerosis) in the lateral column, and may disappear as the lesion clears up.

Buzzard has in several cases witnessed the occurrence of ankle clonus in hysteria, which in one case ceased suddenly, so to speak, on the patient's recovery. He points out, therefore, that the occurrence of ankle clonus must not be regarded as conclusive evidence of a structural lesion of the spinal cord. Buzzard describes an extremely interesting case of a man in whom the patellar tendon reflex was absent on each side during an attack of syphilitic myelitis, which apparently affected a lateral half of the cord. In the limb corresponding to the seat of lesion, the left, it was absent in connection with motor paralysis and muscular wasting, the facts apparently pointing to the arc being "cut" in the anterior horn. In the right limb it was also absent, but in entirely different circumstances, for here the motor power was perfect, and the muscle not wasted, but the cutaneous sensibility was lost. To cause this affection of sensibility, the sensory part of this arc must have been cut. This showed that the lesion, although largely confined to the left half of the cord, encroached a little upon the posterior grey matter of the right half also. In confirmation of this view, Buzzard points to the fact that there was a patch of cutaneous anæsthesia in the neighbourhood of the left trochanter.

Buzzard discusses an interesting point, to which he was the first

to draw attention in 1878—the fact that in tabes dorsalis, with an entire absence of patellar tendon reflex, the muscle is often abnormally irritable to direct percussion. Erb has since (1879) recorded a similar observation. Buzzard finds an explanation of the circumstance in the irritative lesion of the posterior root fibres, and classes it along with the tendency to cramp in sciatica, and clonic spasm of facial muscles in tic douloureux. The case of a patient of his, in whom he had had the infra-orbital nerve stretched for neuralgia, suggested to Buzzard the ingenious and original idea of comparing the tendon reflex in a muscle on each side of the face. He points out that the fifth nerve practically represents the posterior root of a spinal nerve, of which the portio dura is the anterior root, and suggests that in the face we have an opportunity of observation to be found nowhere else, owing to the separation of roots, which in a spinal nerve are bound up in one trunk. He found that he could cause contraction of the zygomaticus major muscle by striking its tendinous origin on the malar bone. In the case in which the sensory root had been stretched (and therefore presumably to a certain extent physiologically impaired), the response on the side of lesion was distinctly less than that on the sound side, although an occasional clonic spasm of the muscles (which had long marked the case—one of severe tic)—seemed to show that the muscle itself was abnormally irritable. The case here appears to Buzzard to resemble strikingly what happens in tabes where tendon reflex is lost in connection with lesion of the sensory portion of the arc, the muscle itself being abnormally irritable. In lateral sclerosis, Buzzard remarks, the converse will often be found to obtain. The tendon reflex will be excessive, whilst the muscles will not respond to a blow, and will require a distinctly stronger faradic current than in health to cause their contraction.

By means of various diagrams, Buzzard suggests explanations of the condition of tendon reflex obtaining in infantile paralysis, protopathic muscular atrophy, lateral sclerosis, deuteropathic muscular atrophy, &c. He makes the important suggestion that the patellar tendon reflex is lost somewhat easily, and that it will often be found wanting when there is but a slight lowering of faradic excitability. Great care is therefore necessary in testing electrically to prevent the error of ascribing the break which he supposes to occur in the nervous arc to a lesion in its sensory instead of its motor portion.

J. Hughlings-Jackson.

Laffont on the Electrisation of the Vago-sympathetic Cord. (*Progrès Médical*, No. 12, 1881.)—The author gives the result of his experiments with reference to the question much discussed lately, whether the stimulation of the sympathetic increases the flow of saliva and the circulation of the labio-buccal region on the same side, owing to the vaso-dilating fibres it is supposed to contain. When on a dog the vago-sympathetic is divided and to the cephalic end a weak current applied, secretion of saliva occurs, and the mucous membrane becomes pale. The latter effect, according to Dastre and Morat, is due to a reflex action, through the sound vagus, upon the heart, or, perhaps, to the excitation of the depressor nerve. But manometric measurements show that there is no diminution in the blood pressure; nor is there diminution, but acceleration of the heart's beats. Hence the pallor observed is due to the direct stimulation of the sympathetic. The dilatation which occurs on the same side after the excitation is purely passive. During the excitation (with a weak current) no vagus-reflex is observed on the same side, but occurs on the opposite side, where the corresponding side is the seat of vaso-dilatation. With stronger currents dilatation appears on both sides, because the powerful vagus-reflex action overcomes the direct excitation of the sympathetic vaso-motors. That the sympathetic in the dog is a purely vaso-constrictor nerve was well shown in a case where the nerve was naturally distinct from the vagus: here no dilatation was observed. The flow of saliva in such experiments is not continuous as long as derived currents through the vagus are carefully excluded.

Rählmann on the Neuro-Pathological Significance of the Pupillary Dilatation. (*Volkmann's Sammlung*, No. 185.)

1. When on illumination the pupil of one eye does not contract, but does so consensually with the other, its optic nerve is still conducting, and there is either oculomotor paralysis, or the movements of the pupil are interfered with by atropia, iritis, atrophy, &c. Blindness of one eye is accompanied with sympathetic reaction when the pupil of the other is made to contract by light.

2. Complete blindness with pupils still reacting (as in uræmia) the lesion lies beyond the corpora quadrigemina.

3. If both pupils react during convergence, both oculomotors are conducting. In paralysis of the pupillary branch of these nerves there is dilatation and immobility, chiefly with complete oculomotor paresis. Unilateral paresis of the pupillary branch is probabably always syphilitic.

4. If both pupils react not to light but to convergence, and if there is some power of vision left, there is a lesion of the fibres of Meynert, between the corpora quadrigemina and the oculomotor nucleus. This condition often obtains in central disease.

5. The medium dilatation depends upon the intensity of the stimulus conveyed to the sympathetic elements of the cervical cord, from the sensory or physical excitations. In weak, nervous people, and in mania, the pupils are often wide. Contracted pupils in maniacal excitement are ominous of subsequent paralysis. Here also signs may occur of rhythmically interrupted action of the sympathetic: pupillary hippus.

6. Narrow pupils characterise all conditions, which depress the cortical functions, especially progressive paralysis of the insane.

7. Narrow pupils are peculiarly common spinal diseases (especially if cervical). In locomotor ataxy the reaction to light is absent, but is normal to convergence. Myosis in this disease is an early symptom.

8. Disturbed pupil diameter along with normal reactions depend upon sympathetic disturbances (megrim, saturnism, exophthalmic goitre, are characterised by irritation and consequent dilatation—contraction, with diminished lid aperture, indicate paresis or paralysis from injury, &c.).

9. Wide pupils are found in impeded respiration, carbonic acid poisoning (acting on the medullary sympathetic centres), Cheyne-Stokes respiration.

10. The same in brain pressure; hydrocephalus.

11. Irregularity shows disturbed sympathetic innervation. Unilateral dilatation with preserved movement is of grave cerebral import; (unilateral oculomotor paralysis is of no serious consequence). Alternating mydriasis, with preserved movement, yields a gloomy prognosis. In addition to this excessive susceptibility to atropia, and persistence of the phenomenon, along with perfect accommodation, are important differential factors. The condition here mentioned is frequent in mental disease and general paralysis.

Regis on La folie à deux. (*Gazette des Hôpitaux*, January 29, 1881.)—Dr. Régis, of the Asylum of St. Anne, recognises two varieties of this disease. First, that in which, of the two individuals who become insane, the one first attacked communicates his insanity to the second who remains on the borderlands of alienation, and may recover on being released from the dominating influence. Second, more complex and rare, where both are subject to the same form

of madness, having been exposed to the same morbid causes and predispositions.

The first division has been fully described by MM. Lasègue and Falret, and may be summarised in the following statements :—

Only one of the two is insane, he having communicated his delusions to the second by a certain moral and intellectual superiority. The individuals have lived in intimate contact. The active agent communicates part of his insanity to the passive; but between the two there is a line of demarcation, the first only being legally insane. Moreover, the latter speedily recovers when removed from the influence of the former.

The second division comprises those cases of true *folie à deux* presenting the following characteristics, as observed in five pairs of cases.

The two individuals become insane simultaneously. They are hereditarily predisposed to mental alienation. They live in intimate and constant contact. The exciting cause operates on both in the same way and at the same time. The mental manifestations resulting are substantially the same; the hallucinations and delusions are the same. Separation of the two is not attended by beneficial results.

Folie à deux may be defined as a monomania, usually of persecution, happening simultaneously to two individuals, the causes of which are—First, hereditary predisposition; Second, intimate and constant contact in daily life; Third, an exciting cause acting on both.

Bourneville and Regnard on Partial Epilepsy. (*Iconographie de la Salpétrière.* MM. Bourneville and Regnard.)—M. Charcot recognises three varieties of this affection: 1, partial epilepsy, with hemiplegia; 2, tonic partial epilepsy, or with contractions; 3, clonic partial epilepsy.

1. The first variety is composed of those who in infancy were seized by convulsions, followed by hemiplegia, and at a variable interval by epilepsy. This paralysis may persist, and a permanent hemiplegia result, or it may almost entirely disappear for a variable period, when it again returns and leaves an irremediable hemiplegia.

The access of this form of epilepsy is thus described. In general there is an absence of the initial outcry. *Tonic period.*—Rigidity of the paralysed side, or predominating in that side. *Clonic period.*—Clonic convulsions almost always limited to the paralysed

side; foaming at the mouth rare; still rarer involuntary micturition. *Period of stertor.*—Very brief, not profound, often absent. *Period of delirium.*—Quick return to consciousness, no epileptic delirium, the subsequent hebetude usually disappearing in a few weeks. After the fit there is a trembling of the paralysed members, and an elevation of the temperature as in ordinary epilepsy. There may be an isolated attack or a series of fits. In a certain number of cases they cease altogether with advancing years.

2. With regard to tonic partial epilepsy, the following are the principal symptoms as observed in one case. A little before the fit the patient complained of throbbing pain in the head, to which was soon added a feeling of constriction at the epigastrium and violent palpitation. In the fit the fingers stiffen and radiate, the face becomes pale, flashes of light pass before the eyes, the eyelids shut spasmodically, the muscles of the right side of the neck contract, there are convulsive movements of the eyelids, the jaws are strongly contracted. The right arm extended and pronated, the hand twisted on the arm rests on the back. The index and middle fingers are outstretched, the ring and little fingers are demi-flexed.

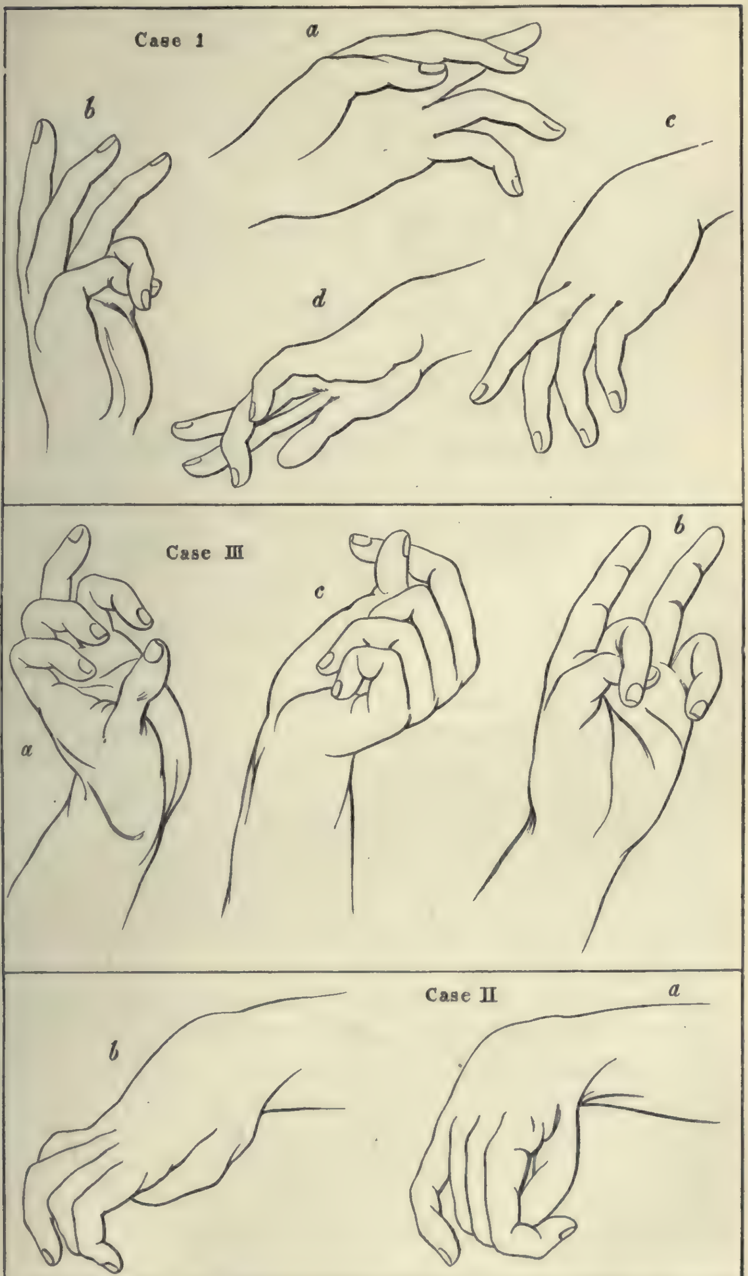
This condition lasts a minute, after which the patient rotates in his chair from left to right, the head being inclined to the shoulder. After two or three minutes these movements are reversed, and he progressively returns to his original position. There is no loss of consciousness.

3. In two cases of partial epilepsy, with clonic convulsions, the spasms were at first confined to a single limb; thence extended to the other limb of the same side, and finally to all four extremities. This form of epilepsy is more serious than the two preceding—one of the two cases having died, the other fallen into dementia.

Debove on a New Method of preparing the Spinal Cord for Microscopic Sections. (*Archives de Neurologie*, July, 1880.)—Dr. M. Debove recommends the following method of hardening the spinal cord for microscopic sections. Place the cord in a 4 per cent. solution of bichromate of ammonia for three weeks. Then in a solution of phenic gum for three days, and for three days more in alcohol. Sections may then be cut with the greatest facility, and placed in water to prevent their curling. They are then immersed in a saturated solution of picric acid for twenty-four hours, and coloured with carmine in twenty minutes—the picric acid acting as a mordant.

A. R. URQUHART, M.D.

CASES OF ATHETOSIS.



To face p. 120. Vol. IV.

BRAIN.

OCTOBER, 1881.

Original Articles.

THE REFLEX INHIBITORY CENTRE THEORY.

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In a paper on Tendon Reflex and Clonus Phenomena, published in August 1880,[1] I had occasion to refer to the increase of the reflex function of the spinal cord which occurs in cases in which the nervous communications between it and the brain have been severed or obstructed. I there stated the ordinary explanation, viz. that in the optic lobes in the frog, and there or thereabouts in the higher animals, there exists an inhibitory centre.

This theory, although useful to us in many ways in the present state of our knowledge, can never be regarded as an exact explanation of the phenomena. Apart from the experimental evidence against it, viz. that stimulation of any afferent nerve will, in the absence of cerebrum optic lobes and medulla oblongata, cause inhibition of reflex action, we must remember that for general reasons an increase of reflex action on removal of the brain is to be expected. In the entire animal the effects of a peripheral stimulus are carried in part to the motor centres in the cord and in part to the brain, in the former resulting in motion, in the latter in sensation. In the decapitated

[1] 'Edin. Med. Journ.,' Aug. 1880.

animal, on the other hand, the nerve channels leading to the brain are cut, and hence the effects of a peripheral stimulus are manifested as motion alone. Of course similar explanations of this increase in the reflex function of the cord on removal of the higher centres, and similar views as to the functions of the cerebrum have been held by many. Schiff[1] is of this opinion, and in support of it he mentions two interesting experiments. One is that in the decapitated newt it will be found that a stimulus applied to the hind legs or tail will produce movements of these parts more and more readily, the more of the upper part of the cord is separated from the lower. The other is, that in the frog the response of the fore legs to peripheral stimulation, by pinching the toes, is increased if the spinal cord be divided in the middle of the back.

That the nature of the influence exercised by the brain on the lower parts of the nervous axis has been recognised is apparent. Thus Lauder Brunton and Carpenter refer to it when they give instances of how the brain can put a check upon the bodily action which some peripheral stimulus would directly produce. For example, Carpenter[2] says: "A man considers himself to have received an affront or injury, which his first impulse is to resent. He restrains himself, however, by a strong effort from immediate action, that effort being the determinate expression of the general conclusion he may have long ago arrived at, that such immediate action is undesirable."

Lewes[3] takes a similar view of the action of the brain, and says: "In spite of the eminent authorities countenancing the hypothesis of a particular set of inhibitory nerves and particular centres of inhibition, I must confess that the hypothesis appears to me inadmissible."

Maudesley[4] says, "the power of the understanding is reflective and inhibitory, being exhibited rather in the hindrance of passion-prompted action, and in the guidance of our impulses than in the instigation of conduct; its office in the individual, as in the race, is, as Comte systematically and emphatically pointed out, not to impart the habitual impulsion but deliberative." Maudesley, however, has certain views as regards the

[1] 'Physiologie des Menschen,' i. p. 201.
[2] 'Mental Physiology,' p. 421.
[3] 'Physical Basis of Mind,' p. 299.
[4] 'Physiology of Mind,' p. 358.

influence of the brain on the lower centres different from those advocated in this paper.

Langendorff in his article on "Reflex Inhibition" in Du Bois-Reymond's 'Archives of Physiology,' 1877, has the following among his conclusions:—

"Die cerebrale, oder wie ich sie lieber nennen möchte, die psychische Form der Reflexhemmung ist eine specifische Function gewisser Theile des Gehirnes, die während des Lebens sich in einer mittleren tonischen Erregung befinden. Es existiren also reflexhemmende Mechanismen. Der Sitz derselben ist nicht ausschliesslich in den Lobi optici zu suchen, sondern auch das Grosshirn nimmt an dieser Thätigkeit Theil.

"Ich betrachte die durch die Reflexhemmung zum Ausdruck gelangende Fähigkeit des Gehirns als einen wesentlichen Factor seiner Intelligenz.

"Damit steht in Uebereinstimmung, dass beim neugebornen Thiere die cerebrale Reflexhemmung fehlt, und dass sie sich erst einstellt, wenn das reifende Bewusstsein die Actionen des Körpers einer Controle zu unterwerfen vermag."

The observer who demonstrated the absence of the cerebral reflex inhibition in the new-born animal was Soltmann, and it is interesting to note in connection with this that according to him stimulation of Hitzig's cerebral areas in new-born animals does not give rise to the usual localised movements.

Finally, Charcot,[1] as the result of a consideration of the physiology, histological examination, and chemical analysis of the brain in the new-born animal, says: "A cet âge, le cerveau n'existe pas encore; au triple point de vue anatomique functionnel et pathologique, c'est un organe indifférent."

So much for the nature of the influence of the brain over the reflex function of the lower parts of the nervous axis; to demonstrate its action many experiments have been made.

Setschenow experimented as follows:—He immersed the foot of the frog in a dilute solution of sulphuric acid, and noted by a metronome the time which elapsed until the foot was withdrawn. He then divided the brain between the optic lobes and

[1] 'Leçons sur les Localisations dans les Maladies du Cerveau et de la Moëlle épinière, 1878–1880,' p. 190.

medulla oblongata, and on repeating the immersion he found the time which elapsed before withdrawal to be diminished.

These results have been corroborated by many other observers, whom it is unnecessary to quote, but it is necessary to mention that Herzen and Langendorff demonstrated that there is also an increase in the reflex excitability of the cord when the cerebral hemispheres have been separated from the parts beneath by a section through the optic thalami. Setschenow had asserted that such an operation caused a diminution of the reflex irritability, but these observers found that when it was performed with a sharp knife, an increase always occurred.

In the experiments which I have made, and to which I now wish to direct attention, I have endeavoured to estimate not the diminution in the time of reflex action which occurs on removal of the higher nerve centres, but the increase in the intensity of the muscular contraction. The arrangements for the experiments were as follows:—

The frog was fixed so that, the tendo Achillis having been divided, the gastrocnemius hung vertically. With the tendo Achillis was connected a weighted lever, and to this lever was attached a pointer, which marked on a smoked surface the height to which the lever rose at each contraction of the gastrocnemius muscle. The weights were 1 or 2 drachms. Stimulation was effected by single induction shocks, the wires (thin ones) from the secondary coil being tied in some cases to the fore legs, in other cases to a fore leg and the hind leg of the side opposite to the one in which the gastrocnemius was detached. The position of the secondary coil was noted, and was, of course, the same all through the experiment, and the wires were not removed from the legs till its conclusion.

The plan of each experiment was to throw in a certain number of shocks at intervals of 30 secs. of such a strength as to produce slight or medium contractions of the gastrocnemius. Secondly, to decapitate or divide the medulla; and thirdly, after two to five minutes' interval, to throw in a similar number of shocks at similar intervals. The height to which the lever rose at each contraction being marked on the smoked surface, any increase or diminution after decapitation could easily be noted.

Before referring in detail to the results of my experiments, I must mention that I made a number of experiments on the decapitated frog to test the influence of time on the reflex irritability of the nerve centres. I did so from a consideration of what George Henry Lewes had noted years ago, and what Goltz and his pupils have demonstrated lately on the dog, viz. that the reflex function is not manifested thoroughly until a certain time has elapsed after the operation. As the result of experiments performed in the method above described, I found that if the decapitated frogs were kept in a moist chamber, irritability remained, and was often increased in periods varying from six to thirty-six hours after decapitation. Let me now direct attention to the experiments detailed in Table I.

TABLE I.

Number of Experi-ment.	Strength of Cur-rent.*	Height to which the weight was raised by 10 contractions, in the intact frog, taken to equal one.	Proportionate height to which the weight was raised by 10 contractions a few minutes after decapitation.	Proportionate height to which the weight was raised by 10 contractions 6 hours after decapitation.	
1	M.	1·6 in. = 1	1·19	..	Wires attached to arm and leg.
2	S.	0·76 „ = 1	1·13	..	
3	S.	1·8 „ = 1	0·95	1·27	
4	S.	1·2 „ = 1	1·10	..	
5	S.	0·9 „ = 1	1·1	..	
6	S.	1·9 „ = 1	0·69	..	
7	W.	0·17 „ = 1	..	2·1	Wires attached to arms.
8	S.	1 06 „ = 1	1·19	..	
9	S.	0·27 „ = 1	..	..	
10	S.	1·16 „ = 1	0·91	1·4	
11	W.	1·6 „ = 1	1·6	..	
12	M.	1·5 „ = 1	1·4	..	

* M = moderate, S = strong, W = weak.

In the first series the weight attached to the right gastrocnemius was two drachms, and the wires from the secondary coil were tied to the right arm and left leg; in the second series the weight was one drachm, and the wires were tied to the arms of the animal.

The first column shows the number of the experiment, the second the relative strength of the induced current employed to stimulate; the third shows the height to which the weight was raised (work done) by ten shocks taken to equal one; the

fourth shows the height in a period of from three to five minutes after decapitation, the fifth (where noted) shows the height six hours after.

A reference to the table will show that in ten out of the twelve experiments a rise after decapitation took place. In two cases this did not occur. These were Cases 6 and 9, in the former of which I was unfortunately unable to test the irritability six hours after the experiment. In seven of the cases the increase was marked within five minutes, and in three within six hours after decapitation.

Two questions now arise. The first is, is there an increase in the intensity of reflex contraction in the frog when the higher nerve centres are removed? On this all observers seem now to be agreed. Cases such as Nos. 6 and 9 in the table occur where no increase can be demonstrated; but these, although somewhat inexplicable, are exceptional.

The second question is, does this increase represent the mechanical effect of the removal of the higher centres, the mechanical effect of the prevention of the passage of nerve force to the optic lobes and cerebrum? I have no hesitation in saying that I believe it does. With all the experiments care was taken during their performance that the conditions affecting nerve and muscular irritability should remain the same, viz. the nature, strength, and mode of application of the stimuli, the load, and temperature. In some cases the muscle was "afterloaded," in others the weight was allowed to bear continually on it, care being taken that the connections of the muscle with the bone, fasciæ, &c., should be the same all through the experiment. The fact that the blood-supply to the nerves and muscles will be stopped, owing to the bleeding which occurs on decapitation, will not affect their irritability in any way.[1]

It might be asserted, however, that the increase of irritability is due to the effects of the decapitation on the excitability of the parts below. We know that a slight increase of irritability follows for a short time section of a nerve-trunk, that strychnia increases greatly the irritability of the cord, and that a somewhat similar condition can be brought about by

[1] See Langendorff, ibid. p. 111, note.

the electrotonic state, and by some nerve irritation, as in Brown-Sequard's epileptic guinea-pigs.

But the explanation of the increase is, I think, not to be found in any of these; the fact that in most cases it makes its appearance in a few minutes, and lasts for hours after the decapitation, showing that it is not a temporary but a permanent condition.

We might also on first consideration suppose that the increased patellar tendon reflex in lateral sclerosis might be due to the sclerosis increasing in some way the irritability of the motor parts below, just as we may suppose that in Locomotor Ataxy the characteristic pains are caused by the sclerosis below increasing the irritability of the sensory parts above, and we might suppose that the marked increase in the reflex function of the cord, which Goltz has lately described as occurring in animals weeks or months after section, and scarcely less marked examples of which we have all seen in man at similar periods after disease or injury of the cord, are due to a like structural change in the nerve tracts and centres. While allowing, however, that the irritability of the nerve centres may be influenced by nutritive changes, sclerosis, &c., of certain tracts, I have no hesitation in asserting that the increased reflex in disease, as in the frog experiments, is in great part due simply to the concentration of the effects of the stimulus as mentioned in the first part of this paper.

In support of this theory are, I think, those cases of temporary hemiplegia due to cortical lesions in which there are exaggerated knee jerk and foot clonus, disappearing with the paralysis. Through the kindness of Dr. Brakenridge, I had the opportunity of seeing one such case in his wards in the Royal Edinburgh Infirmary last winter, and a particularly interesting case has been reported by Dr. Hughlings-Jackson.[1] Here, as the symptoms lasted only a few hours, there could be no sclerosis, and I believe that the increased reflexes were simply due to the condition of the cortex rendering it incapable of receiving a stimulus and so causing a concentration of nerve force in the parts below. Hughlings-Jackson, however, gives another explanation.

[1] 'Med. Times and Gazette,' Feb. 1881.

But evidence in support of this concentration theory is, I think, to be obtained by experiment. A closer consideration of the table will show that the increase of contraction after decapitation in the second series is greater than in the first. The explanation of this we may suppose to be that when the wires from the battery are connected with the forelegs of the frog, there will be a greater tendency for the effects of the stimulus to pass to the higher centres than when they are connected one with a fore leg and the other with the hind leg of the opposite side. On this theory we should expect that when the reflex contraction is produced by an electric stimulus sent through the fore legs, its intensity should not be so great as that of the contraction produced by sending the current from a fore to a hind leg, or through the hind legs. On the other hand, however, we should expect that the proportionate increase of the contraction after decapitation should be greater where the stimulus is sent through the forelegs, inasmuch as a greater portion of the nerve impulse generated by it will be kept concentrated in the cord. To obtain further evidence on this point, I performed the experiments detailed in Table II.

TABLE II.

Number of Experiment.	Strength of Current.	Proportionate height to which the weight was raised by 10 contractions in the intact frog.		Proportionate height to which the weight was raised by 10 contractions a few minutes after decapitation.	
		When the wires were attached to the arms.	When the wires were attached to one arm and one leg.	When the wires were attached to the arms.	When the wires were attached to one arm and one leg.
1	M.	1·5 in. = 1	0·9	1·03	0·9
2	M.	1·27 „ = 1	0·8	1·2	1·4
3	M.	0·86 „ = 1	1·2	1·4	1·3
4	M.	0·7 „ = 1	1·2	1·7	1·6
5	M.	1·9 „ = 1	1·1	1·2	1·2
		Wires attached to the arms.	Wires attached to the legs.	Wires attached to the arms.	Wires attached to the legs.
6	M.	1·7 in. = 1	1·4*	1·2	1·5*
7	M.	1·7 „ = 1	1·03*	1·2	1·1*
8	W.	0·9 „ = 1	1*	1·05	1·1*
9	M.	1·6 „ = 1	2*	1·7	2·1*

* Contraction to making and breaking induction shocks.

In both series the weight was one drachm. In the first series the first column represents the number of the experiment, the second the strength of the current, the third the height to which the weight was raised when the ten shocks were sent through the arms, taken as one, and the fourth the same when the shocks were sent by the wires attached to a fore and a hind leg. The fifth and sixth columns represent the heights attained by contractions induced in the same way, three or four minutes after decapitation. As regards series 2, the only explanation further required is that the contractions, the height of which are marked in the fourth and sixth columns, were produced with the wires attached to the hind legs immediately below the knee-joints.

An examination of the table shows that in the first series the contractions produced by stimuli sent through the fore legs were in the intact frog less in three cases out of five than those produced by sending the stimuli through a fore and a hind leg, in two cases they were greater. In the second series, in three out of four cases the contractions induced by stimulations sent through the forelegs were less than when sent through the hind legs; in one case they were similar in intensity. Both series show that the increase after decapitation was more marked when the forelegs were stimulated, the exceptions being No. 2 in the first, and No. 3 in the second series.

These results therefore agree in the main with what we on theoretical grounds assumed, and are, I think, in favour of the view that the increased contraction, which occurs, either on removal of the higher centres or where from any cause nerve impulses are prevented from passing to them, is due to a concentration of nerve force. But granting that this increased contraction is due to this extra amount of nerve force being sent to the muscles, can we form any estimate as to its amount? We have just seen that it is greater when the stimuli are sent through the arms than through the legs, and we have offered a possible explanation of this. But when we reflect on the many conditions by which the muscle and nerve irritability of the frog may be influenced, and consider that the experiments are for such a purpose few in number, we shall not attempt to form any very exact estimate.

In Table II. the average increase in height after decapitation when the stimuli were sent through the forelegs, was ·29, the least being ·03 and the greatest ·7; whilst when the stimuli were sent through the fore and hind leg, as in the first series, or through the hind legs, as in the second series, the average increase was ·17, the least being 0 and the greatest ·6.

But whatever value we may ascribe to those numbers, it is satisfactory to be able to remark that they are to a certain extent at least corroborated. Thus Langendorff, experimenting after the method of Setschenow on the diminution in time of the manifestation of reflex action on removal of the higher centres, says that the "reflex strength," i.e. the rapidity with which the response follows a peripheral stimulation, is inversely proportional to the time, and gives the following as his first experiment.

Versuch I.

Linkes Bein.		Rechtes Bein.
9		9
7–8		8
	Säure bedeutend Verdünnt.	
		Schnitt durch die rechte Hälfte der Med. obl. dicht hinter dem Cerebellum.
14		18–19
10–11		18–19
12		15
10		17–18

In this experiment Langendorff, by dividing the right half of the medulla oblongata, severed a nervous connection (the nature of which we shall say a little more about by-and-by) between the left half of the cord and the higher centres. He thus increased the reflex irritability of the left leg, as shown by the shorter periods which elapsed before reflex contractions took place. If now we remember that the reflex strength is inversely proportional to the reflex time, the consideration of his results will, I think, convince us that our results are only such as might have been *à priori* expected.

But now an important consideration presents itself. If this increase in the intensity of muscular contraction, which takes place when the higher centres (medulla oblongata, optic lobes, cerebrum) are removed, is brought about by the nerve force which would otherwise have passed to them being directed to

the muscles, and if in the normal condition the passage of this nerve impulse to those centres will excite their several functions, it follows that our experiments may be looked upon as affording what may be called a "mechanical equivalent" of their collective functions. It follows, further, that if we in a similar manner test and compare the strength of the reflex contraction before and after the removal of the cerebral lobes only, we may obtain a "mechanical equivalent" of their individual functions. I have performed this experiment several times, but was only fairly successful in two cases. In the first, the rise after removal of the cerebral lobes was 0·7; in the second, it was ·1; and on consideration of these experiments I must mention that I believe that the rise in the first case was too great, inasmuch as when the frog was experimented on before removal of the cerebral lobes, it had not quite recovered from the effects of the removal of the skull-cap. This of course is only a hypothesis, and I regret that I have no other experiments bearing on this point to adduce.[1] We should, however, expect to find, if our theory be correct, that the increase in the reflex contraction is not so great after removal of the cerebral lobes as after removal of the optic lobes and medulla as well.

In stating that these experiments may afford a mechanical equivalent of the functions of the higher parts of the nervous system, many important points require to be considered.

In the first place, we must remember that a stimulus of some sort is required to excite function in these higher centres, so that, supposing that they are removed, or their power of response to a stimulus is destroyed, a something will be kept in the cord which would otherwise have left it. Of course this stimulus might bear no proportion to the amount of energy which it will liberate in those higher centres, just as an explosion of tremendous magnitude may be produced by the slightest pressure on a hair-trigger.

We must, in the second place, remember that the mechanical effects will vary with stimuli of the same nature and strength,

[1] Owing to legislative enactments in this country, the experiments entailing vivisection were performed on the Continent, under rather disadvantageous circumstances, at the beginning of last summer session.

according to the areas to which they are applied. Thus in our experiments we have seen that the increase after removal of the higher centres was greater when the stimuli were applied to the fore than to the hind legs. We have given a probable explanation of this, and need no more than mention the well-known experiments of Pflüger on the directions in which nerve impulses, generated as the result of peripheral stimulation, are transmitted.

We must remember in the third place, also, that stimuli of the same nature, of the same strength, and applied to the same parts, will produce very different effects in different individuals, and in the same individual at different times. Very good examples of this in different individuals are given by Lauder-Brunton in his article on "Inhibition, Peripheral and Central," in the 'West Riding Asylum Reports for 1874,' vol. iv. Thus a stimulus applied to the peripheral terminations of the optic auditory or cutaneous nerves, which in the boy will be almost entirely expended in producing muscular contraction resulting in a blow, "may, by passing to the brain, lead to action by-and-by, as in a Corsican vendetta, where the avenger pursues his intended victim for years until a favourable opportunity occurs, and by a thrust of his dagger he satiates his thirst for vengeance. Or they may excite ideas of the sinfulness of revenge and the advantages of peace, and thus lead to pacific actions. Or, finally, they may be nearly effaced by the effects of other stimuli of a different nature, arising from the pressure of daily work and daily care, and lead to no action at all, though it is probable that they always leave an alteration more or less slight in the ideational centres."

As an example of stimuli of the same nature, of the same strength, and applied to the same part, producing in the same individual different effects at different times, no better can be quoted than that given by the late George Henry Lewes.[1] In explaining the action of stimuli on respiration, he mentions that the "external local application," which in the new-born child acts as a stimulus to the respiratory and motor centres, will in later years "be freely used as a stimulus to virtue or learning."

[1] 'Physiology of Common Life,' p. 403.

In addition, we must remember that the increase in the extent of the contractions which takes place after removal of the higher centres, will not represent the total amount of the nerve force which, in the intact frog, would have gone to stimulate these parts, inasmuch as there is an overflow of this nerve force to other muscles which we cannot estimate, and which may not be proportionately equal before and after the removal of these centres.

I need therefore say no more as regards the possibility of obtaining a precise mechanical equivalent of nerve or brain-work; but there are certain facts which seem to corroborate my experimental work, in denoting that it must be very high, and these I shall now proceed to discuss.

It has been estimated, for example, that in man the proportion of the weight of the brain and spinal cord to body-weight is about 1 to 40, and that the amount of blood circulating at any moment in these parts is about one-fifth of the total amount of blood in the body. This shows that the molecular decomposition in nervous tissue is enormously active; this is further shown by the instantaneous effects of deprivation of blood supply, e.g. the syncope which follows compression of the carotids, the paraplegia which follows ligature of the abdominal aorta.

Remembering this, and the frog experiments, we can believe the phenomenon related by Carpenter[1] that Braid (the Manchester Surgeon and investigator of Hypnotism), in his presence, enabled a man, so remarkable for the poverty of his physique that he had for many years scarcely ventured to lift a weight of 20 lbs., to take up a weight of 28 lbs. on his little finger and swing it round his head with the greatest apparent ease. Carpenter says "neither Mr. Braid nor his son, both of them powerful men, could do anything like this, and I could not myself lift the same weight on my little finger to more than half my own height."

This patient was, of course, in the hypnotic state, and this and many other of the hypnotic phenomena of the intensifying of the senses of hearing, sight, smell, &c., have been explained as being due to a like concentration and consequent intensification of nerve force.

[1] 'Mental Physiology,' p. 606.

But, granting that nerve force has a very high mechanical equivalent, to enable us better to understand examples of this, let us endeavour to discover how a concentration of nerve force and consequent intensification can be brought about. It appears that this can be done in either or both of two ways; first, by preventing overflow of nerve force from the nerve tract involved to other tracts; and, secondly, by preventing interference with the nerve force in the nerve tract involved by overflow into it from other tracts.

In every case we may, I think, suppose that both of these conditions may act. For instance, the increased reflex action in the decapitated frog, in paraplegia, lateral sclerosis, &c., may be assumed to be due to the prevention of overflow from certain tracts in the cord; but we must not forget that in the intact frog, or in the healthy individual, the slighter reflex response may have been due to interference with the nerve force in the lower part of the cord by nerve impulses passing downwards from the brain.

Similarly with the hypnotic experiments, it is impossible to say in such a case as that detailed by Carpenter how much of the great increase of nerve force was due to either of those conditions acting separately.

But we do not require to look to frogs, hypnotic patients, or paraplegics to assure ourselves of the value of concentration of nerve force. We all know how the action of stimuli—visual, auditory, cutaneous—interferes with study, and conversely how the working out of a difficult problem renders us for the time incapable of perceiving all ordinary stimuli. The staggering gait and incoordinate purposeless movements which occur in the child when he is learning to walk may be looked upon as being in part, at least, due to a want of concentration, for here the various stimuli necessary for equilibration—cutaneous, muscular, labyrinthine, visual, visceral—are probably carried to other areas, as well as those intended to receive them, and from those areas the motor-nerve impulses are carried to muscles other than those required to contract. As the result of practice, however, certain definite nerve tracts are channelled out; no useless escape of nerve force takes place, and the function is performed perfectly. Similarly in learning to read, to play a piece of music; we all know that such

actions can be best performed when they are done, as the expression is, "without thinking."

Our theory also enables us to understand the difference in the manifestations of a developed and an undeveloped intelligence. As Herbert Spencer[1] says, "The brain of the uncultivated man, as compared with that of the cultivated man, must be one in which the routes taken by nervous discharges are less numerous, less involved, less varied in the resistances they offer; one, therefore, in which the number of ideas that can follow a given antecedent is smaller, and the degrees of strength with which they can present themselves are fewer; one, therefore, in which the possibilities of thought are more limited, and the balancing between alternative conclusions less easy. This is the reason that ignorant people generalise hastily, and adhere obstinately, to the erroneous conclusions based on their scanty experiences; while the highly instructed man is able to keep his judgment undecided, waits for more evidence, contemplates other possible inferences than the one he is inclined to draw, and is ready to abandon or qualify his conviction when he discovers facts at variance with it."

No stronger argument in favour of the concentration theory can be brought forward than the constantly observed association in individuals of ignorance and obstinacy.[2]

Assuming now that this increase in the reflex function of the cord which ensues on the removal of the higher centres is due to the obstruction or cutting through of nerve channels, and consequent concentration, our next subject is to consider more in detail what those channels are.

The first idea which is here likely to occur is that they are sensory, inasmuch as we stated at the beginning of this paper that the impulse generated in the cord by peripheral stimulation is in part conducted to the brain, to produce consciousness, and in part to the muscles, to produce motion.

The clinical evidence on this point is very conclusive. Carpenter[3] relates two cases recorded by Dr. William Budd,

[1] 'Principles of Psychology,' vol. i. p. 582.

[2] Compare Letourneau's 'Sociology,' Book III. chap. i. "The Reflex action according to Race and Civilization."

[3] 'Mental Physiology,' p. 70.

in which there was more or less complete paralysis of motion and sensation in the legs as the result of spinal lesion in the dorsal region. The reflex response of the muscles of the legs to pricking, pinching, was greatly increased even when those stimulations were felt by the patient, but the stimulations which were most efficacious in producing reflex contraction were those of which "no consciousness whatever was experienced."

But the sensory channels are not the only ones, obstruction of which leads to increased reflex action. In lateral sclerosis of the spinal cord, an increase of the reflex irritability (both skin reflex and knee jerk) is common, and here, since there is not necessarily any diminution of the cutaneous sensibility, we may suppose that the motor channels alone are obstructed. Again, Langendorff has demonstrated that one side of the brain exercises its so-called "inhibitory" influence over the opposite side of the body, and that these "inhibitory" fibres cross in the medulla oblongata. This corresponds to the paths followed by the motor fibres, and corroborates clinical observation in lateral sclerosis.

But since obstruction of motor fibres in the cord causes, by corresponding concentration, increased reflex action in the parts below, we have here additional evidence for the view that such fibres carry centripetal as well as centrifugal impulses. That nerves can conduct in both directions is now admitted, and many observations could be quoted on the point. The one mentioned by Kühne, that if one terminal twig of a forked motor nerve be stimulated, activity is induced in the other terminal twig, provided the common trunk be intact, and that hence the fibres of the former must have conducted in a centripetal direction, seems, however, specially applicable.

Finally, since the grey matter can conduct both motor and sensory impulses, we may suppose that section or obstruction of it alone would lead to increased reflex action of the other parts of the cord.

THE LOCALISATION OF ATROPHIC PARALYSES.

BY DAVID FERRIER, M.D., F.R.S.

(Continued from page 232.)

Among other clinical cases illustrative of the grouping of functionally related muscles indicated by experiment, there are some reported by Remak himself, and others cited by him in his memoir already quoted.[1]

In Obs. II. the biceps, brachialis anticus, and supinator longus were affected on both sides, and on the left, also, the extensors of the fingers and wrist.

In Obs. III. there was a similar combination of "the upper-arm type" with the extensor indicis on both sides, and some affection also of the extensor carpi radialis on the left.

In a case reported by Bernhardt,[2] there was well-marked atrophic paralysis of the "upper-arm type," with degeneration also of the extensor communis digitorum, and minimi digiti on the left side.

These cases illustrate the combination characteristic of the fourth and fifth cervical roots. In all probability the other muscles, synergic with those actually mentioned, were also affected more or less, in accordance with the cases I have myself reported above.

It seems less common to have limited poliomyelitis of the lower cervical and first dorsal type, than of the upper portion of the cervical enlargement.

But Seeligmüller[3] reports a case of infantile paralysis of this kind, in which the interossei and thenar muscles, and flexors of the wrist and fingers were affected, while the extensors of the wrist and basal phalanges were intact, giving

[1] Archiv f. Psychiatrie, 1879. [2] Ibid. viii. p. 780.
[3] Jahrb. für Kinderheilk. N. F. xiii. p. 353.

rise to a "main à griffe" like that of hypertrophic cervical pachymeningitis.

The lesions causing atrophic spinal paralysis of the lower extremity are to be sought for in the region included between the origins of the third lumbar and second sacral nerves, and the special combinations in the segments corresponding to the several roots as described above (p. 226).

Limited poliomyelitis anterior occurs in the lumbar as in the cervical enlargement, more especially in children, but there are greater difficulties in the way of a satisfactory exploration of the individual muscles. But that the grouping of the muscles affected together has no relation to the peripheral nervous supply, is sufficiently clear.

The escape of the sartorius in cases where the other muscles supplied by the anterior crural nerve are affected is a fact which has been noted by Erb, Remak, and others. We can account for this by reference to the results of stimulation of the third lumbar root. The sartorius is more especially in relation with the ilio-psoas, or flexors of the thigh on the pelvis. Though the extensor cruris is also represented in this root, it is again represented in the fourth lumbar, with the extensors of the thigh and of the first metatarsal bone.

The extensor cruris may be therefore severely affected, while the sartorius, if affected at all, may not show discoverable signs of impairment. Here, as in other cases, the healthy fibres might so outnumber any degenerate ones that the electrical reactions might fail to detect signs of degeneration.

Remak is disposed to correlate the tibialis anticus with the extensor cruris, and he adduces in support some cases of the conjoint affection of these muscles, the other muscles supplied by the peroneal nerve retaining their contractility.

In the ordinary movements of the lower extremity, however, the action of the tibialis anticus—raising the inside of the foot—seems to be associated more with the flexion of the leg, as in the action necessary to clear the foot of the ground preparatory to the swing forward of the limb in walking, and still more evidently in climbing a stair. Without the action of the tibialis anticus the point of the foot would inevitably catch.

The combination of movements excited by stimulation of the fifth lumbar root in the monkey is that involved in bringing the foot towards the trunk, as when the animal scratches its flank or abdomen with its toes. There is, therefore, a very close resemblance between the action and that of the fifth cervical. Here the hand is brought to the mouth, by abduction of the humerus, flexion and partial supination of the forearm, and clawing of the fingers; and similarly by the fifth lumbar the thigh is abducted, the leg flexed, and the foot so set as to bring the toes in claw fashion towards the middle line.

The tibialis anticus would thus correspond very nearly to the supinator longus, an analogy which would not hold if it were regarded as functionally associated with the extensor cruris.

The association of the extensors of the thigh, extensor of the leg, and extension of the first metatarsal bone by the action of the peroneus longus, the result of stimulation of the fourth lumbar, is necessary for the full rigid extension of the limb backwards, and forward propulsion of the body in walking, while the opposite limb swings forward. Theoretically, we might expect to find cases of limited poliomyelitis of the fourth lumbar type in which some of the gluteal muscles, extensor cruris and peroneus longus should be affected together. A case of this kind—more or less complete—has been recorded by Buzzard.[1] The patient, a girl aged 16, exhibited in the right leg a total absence of response to the faradic current in the quadriceps extensor cruris and peroneus longus, while the anterior tibial group responded to strong currents. The gluteal muscles are not specially alluded to.

This case may be put against those cited by Remak as apparently establishing a correlation between the extensor cruris and tibialis anticus. But even apart from this, the clinical material at present existing for the functional correlation and spinal localisation of the muscles affected by atrophic paralysis in the lower extremity is so indefinite, that trusting to this alone may readily cause mere coincidence or juxtaposition to be mistaken for interdependence or causal relationship. We require more numerous observations, and a more thorough

[1] 'Medical Press and Circular,' Feb. 16, 1881.

exploration of all the muscles of the lower extremity, before we can go much beyond mere plausible conjectures from a purely clinical standpoint.

Saturnine Paralysis.—Though the phenomena of chronic lead-poisoning have been the subject of more numerous investigations, clinical, pathological, and experimental, than almost any other neuropathy, it cannot be said that any of the theories as to its nature, whether myopathic, peripheral, or spinal, have succeeded in giving an explanation at all satisfactory of the characteristic drop-wrist, which is such a striking feature of saturnine paralysis. So prominently has this special symptom occupied attention, that it has led to theories as to the mode of action of lead of a very narrow or mechanical character, to the neglect of other symptoms which they entirely fail to account for. The explanation of lead-paralysis by a particular affinity of lead for the muscles, which was apparently established by the researches of Gusserow,[1] was overthrown by the subsequent researches of Heubel.[2] Heubel found that the muscles, instead of yielding the greatest quantity of lead, contained least of all, when compared with the bones, kidneys, liver, brain, and spinal cord.

The investigations of Bernhardt,[3] with a view to determine a relative difference in the amount of lead contained in the paralysed extensor communis and the normal supinator longus, were entirely negative—researches which, the author states,[4] he would not now think of undertaking in the present state of our knowledge on this question.

It is almost unnecessary to mention the hypothesis advanced —now abandoned—by Hitzig, to account for the special affection of the extensors, by a mechanical stagnation of the lead-containing blood in them, owing to defective venous efflux. A similar mechanical explanation advanced by Henle may be passed by without further comment.

The researches of recent years have considerably narrowed the questions at issue, and the points now in debate are, whether lead acts primarily on the peripheral nerves or on the

[1] Virchow's 'Archiv,' 1861.
[2] 'Path. u. Symptom. d. Chron. Bleilähmungen,' 1871.
[3] 'Archiv für Psychiatrie,' iv. p. 616. [4] Ibid. vii. p. 325.

spinal centres. A myopathic origin is at present maintained by few, if any, but Friedländer.[1]

But even if we should adopt either the spinal or the peripheral origin, the question arises, why should the spinal nuclei, or motor nerves of the extensor communis digitorum, be of all others specially attacked by lead.

Before attempting a solution of this question, it will be well to take a general survey of the clinical phenomena which have been met with as the result of lead-poisoning. From these it will be seen that the paralysis of the extensors of the fingers and wrist is but a very small and comparatively insignificant fact, though it has largely overshadowed the others. In the classical work of Tanquerel des Planches,[2] and in numerous special memoirs by various other authors, we find enumerated among the effects of lead, various forms of *encephalopathy*, psychical or epileptiform, with intense headache, amaurosis, and optic neuritis, simulating cerebral tumours; *hemianæsthesia*;[3] *severe pains* in various parts of the body, which often differ in no respect from those of locomotor ataxy;[4] *anæsthesia*, irregularly distributed, and varying in degree;[5] *paralytic affections*, not merely of the extensors of the forearm, but of the whole limb, and also of the lower extremities, partially or wholly; *tremors; ataxy*;[6] *aphonia; spasmodic asthma*, &c.; all tending to show that the action of lead on the nervous is of an all-pervading character, not to speak of its direct action on the blood corpuscles, intestinal canal, blood-vessels, kidneys, and liver.

Post-mortem investigation of the brain, spinal cord, spinal roots, and peripheral nerves, has sometimes been entirely negative, no lesions having been discoverable though the paralytic symptoms were well marked.

But, on the other hand, a considerable number of observa-

[1] Virchow's 'Archiv,' lxxv.

[2] 'Traité des Maladies de Plomb,' 1339.

[3] De Cours, Thèse, 1875, 'L'Hémianesthésie Saturnine;' Renaut, 'L'Intoxication Saturnine Chronique,' 1875 (containing several observations of this nature by Vulpian and Raymond); Manouvriez, 'L'Intoxication Saturnine,' 1874.

[4] Compare Tanquerel's description, vol. i. p. 506.

[5] Beau, 'Rech. sur l'Anesthésie Saturnine, Archiv. Gén. de Médecine,' 1848; Manouvriez, op. cit.

[6] Renaut, op. cit. Obs. xviii.

tions of a positive character have been published of late years, in which morbid changes have been found in the brain, spinal cord, and peripheral nerves, as well as in the muscles.

The paralysed muscles have been generally found to be pale, atrophied, the striation diminished, and the nuclei and connective tissue increased.

In lead encephalopathy the brain has occasionally been found only yellowish or slightly altered in density. But in a case of lead-paralysis, with encephalopathic symptoms, recently recorded by Monakow,[1] the condition of the brain was such as is generally met with in general paralysis, viz. atrophic degeneration, more especially in the frontal and parietal regions and at the base. In the atrophic regions the vascular spaces were dilated; there were accumulations of pigment, hyperplasia of the neuroglia, altered nerve-cells, &c.

In the spinal cord sometimes no morbid appearances have been discovered,[2] but Vulpian and Raymond[3] found in one case that the spinal roots and nerves were normal, but in the cervical enlargement a number of cells in the anterior cornua were atrophied, shrivelled, pigmented, and vacuolated, with other indications characteristic of anterior poliomyelitis.

Monakow found in his case[4] morbid changes in the middle of the cervical enlargement, viz. hæmorrhages, vascular changes, atrophy of the ganglion cells, along with sclerosis of the posterior horn on the left side, corresponding with analgesia of the left side and hyperæsthesia of the right.

In the spinal nerves a granular degeneration of the myelin, with proliferation of the interstitial connective tissue, has been described, in the radial nerve, by Lancereaux, Gombault, Monakow, &c. In a case recently published by Moritz,[5] the intra-muscular nerve fibres of the paralysed extensors were thickened, the nuclei multiplied, and the axis-cylinders more or less degenerated. A careful examination of the cervical enlargement by Dreschfeld revealed nothing abnormal, and

[1] 'Archiv für Psychiatrie,' x. 1880.
[2] See Moritz and Dreschfeld, Eisenlohr, Friedländer, &c.
[3] Vulpian, 'Maladies du Système Nerveux,' 1879, p. 158. [4] Supra cit.
[5] 'Journal of Anatomy and Physiology,' Oct. 1880.

the rest of the cord examined by Moritz was likewise negative.

A similar condition of the intra-muscular nerves, with absence of morbid change in the spinal roots and spinal cord has been described by Eisenlohr.[1]

Friedländer also could detect no morbid appearances in the spinal cord in his case,[2] though there was marked degeneration of the intra-muscular nerves extending into the large nerve trunks.

Experimental investigation in the lower animals, as to the effects of long-continued administration of lead-salts, has led to results of considerable value.

Vulpian developed in a dog paralysis first of the hind limbs and next of the fore limbs by repeated doses of carbonate of lead. After death, indications of myelitis and degeneration of the multipolar cells, and alterations in the nerve-fibres were discovered.

More recently Gombault[3] has described the effects of chronic poisoning with lead on guinea-pigs. Though no special paralytic symptoms were developed, yet after death the nerve-trunks of the limbs exhibited morbid appearances which he has termed segmentary periaxillary neuritis. At various points in the nerve-fibres there was degeneration of the myelin with retention of the axis cylinder in various stages of progress or restoration. In the spinal cord, also, were lesions such as are found in anterior poliomyelitis, the nerve-cells being vacuolated, and the neuroglia and vascular walls showing indications of inflammatory hyperplasia.

We have, therefore, a large number of observations, clinical and experimental, in favour of actual morbid conditions induced by lead in the brain, spinal cord, and peripheral nerves. With the exception of Gombault's experimentally-induced periaxillary neuritis, which affected the nerve-trunks apparently generally, the attention of pathologists seems to have been concentrated on the musculo-spiral and its branches to the extensor digitorum; but the demonstration of morbid changes in this nerve does not exclude

[1] 'Deutsch. Archiv f. klin. Med.' xxvi. p. 543. [2] Loc. cit.

[3] 'Archives de Neurologie,' Nos. 1 and 2.

the existence of similar appearances more or less marked in other nerve-trunks. And we have good reasons for supposing that such is actually the case. Even when the extensors of the fingers are alone paralysed, there is a general muscular enfeeblement which has frequently been noted by others, and which I have invariably found in cases of saturnine paralysis under my own care. A general subnormal electric excitability has also been observed in some cases. In a case in which there was drop-wrist of the left arm, Buzzard[1] found diminished faradic excitability generally, in muscles which functionally were apparently normal. And the tendency of severe saturnine poisoning to cause paralysis, not merely of the extensors, but of numerous other muscles, both in the upper and lower extremities, taken with facts like these, indicates that underlying the pathology of drop-wrist there is something more than mere mechanics or a specific influence on the radial nerve or its spinal nucleus.

It is a matter of everyday observation that in ordinary hemiplegia the extensors of the fingers are the last to regain their power in the process of recovery, so that if after general loss of power in the limb, the patient can extend his basal phalanges, the recovery of the other movements may be taken as a matter of course. It is the same with general cervical anterior poliomyelitis. In a case of this kind, under the care of Hughlings-Jackson and myself, the extensors of the fingers and wrist still remained paralysed after all the other movements had been regained, if not of their normal strength.

So also in peripheral musculo-spiral paralysis, the extensors of the fingers are the last to recover. In a case I saw recently all the movements had been regained except extension of the middle and ring finger, which appear to be the weakest of all.

A similar condition obtains in the lower extremity as regards the peroneal muscles and extensors of the toes.

But it is not only in the limbs that the extensor or analogous muscles seem to be thus more especially enfeebled. In a highly suggestive paper,[2] Dr. Semon has shown that there is a special proclivity of the abductor muscles of the larynx to become paralysed, before the adductor muscles, or

[1] 'BRAIN,' No 1. [2] 'Archives of Laryngology,' vol. ii. No. 3, July 1881.

even exclusively, in cases of central or peripheral disease of the vagus, spinal accessory or recurrent laryngeal nerve. He gives numerous cases illustrative of this affection, and at the same time points out that there is no case on record in which similar disease has ever led to isolated paralysis of the adductor muscles.

Of great significance in this relation also is a fact cited by Renaut,[1] that according to the observations of Günther, Gurlt and Hertwig, horses employed in lead works suffer from lead poisoning, and a laryngeal affection in which it has been found that certain of the fibres of the recurrent laryngeal nerve are atrophied, and the posterior crico-arytenoid muscles in a state of fatty degeneration.

Other observations of a different character, but also bearing on this question, have been recently published by Onimus.[1]

He finds that after the brain and spinal cord, the great nerve trunks lose their excitability after death; then the nerves to the extensor muscles, and lastly those of the flexors, which may remain excitable from two to four hours after death. In the lower extremity the peroneal nerves lose their excitability before the tibial.

The extensor muscles also lose their excitability before the flexors, both in the upper and lower extremities.

These facts all clearly point to the conclusion that the extensor and abductor nerves and muscles have less vital resistance and are sooner exhausted than the flexors. We may argue from this that a generally enfeebling cause will show itself first prominently in the extensors.

The physiological strength of a muscle does not seem capable of being accurately estimated by its mere sectional area. The degree of innervation must also be reckoned. Without considering the mere quantity of muscular fibre, we may roughly estimate the relative physiological strength of the various muscles and muscular groups which act together, including their innervation, by the force they are capable of manifesting during strained volitional effort.

[1] Op. cit. p. 49.

[2] "On the Excitability of the Nerves and Muscles after Death" ('Journal de l'Anatomie,' No. 6, 1880).

With the view I have made a comparative estimate of the strength of the various movements of the trunk and limbs in an individual of average muscular strength, without special development of particular muscles. The weight was placed in all cases at the point of best advantage to the muscle, as near its insertion as convenient to avoid loss by leverage, and the number of pounds raised by forced effort registered.

HEAD AND TRUNK.

	lbs.
Retraction of head	25
Bending head	22
Straightening trunk	90
Bending trunk	65

UPPER EXTREMITY.

Elevation of shoulder	80
Retraction of upper arm	65
Forward extension of upper arm	40
Adduction of arm	40
Abduction of arm	35
Outward rotation of arm	10
Inward " "	10
Flexion of forearm	65
Extension of "	30
Flexion of wrist	22
Extension of wrist	22
Pronation of "	18
Supination of "	15

	Prox. Phal. lbs.	Middle Phal. lbs.	Distal Phal. lbs.
Flexion of thumb	20	—	14
Extension of "	8	—	4
Abduction of "	14	—	—
Opposition of "	15	—	—
Adduction of "	7	—	—
Flexion of fingers:—			
1st finger	8	10	6
2nd "	9	11	8
3rd "	9	11	8
4th "	7	9	6
Extension of fingers:—			
1st finger	4·5	3	– 2
2nd "	5	3	– 2
3rd "	5	2·5	– 2
4th "	4·5	2·5	– 2

Extension of four basal phalanges together	10 lbs.
Adduction and abduction of fingers (average)	5 "

LOWER EXTREMITY.

	lbs.
Extension of thigh	80
Flexion of "	60
Abduction of "	60
Adduction of "	50
Flexion of leg	50
Extension of leg	40
Plantar flexion of foot	80
Dorsal " "	32
Raising inner side of foot (tibialis anticus)	26
Raising outer " " (Peronei)	23

	Prox. Phal. lbs.	Distal Phal. lbs.
Flexion of big toe	18	18
Extension of big toe	13	6

	lbs.
Flexion of outer toes together	15
Extension of	10

The relative strength of the various movements stated above is strictly applicable only to volitional innervation which might not correspond with the relative innervation by the spinal centres. But that a similar proportion obtains also in respect to spinal innervation is shown by the position which the trunk and limbs assume in tetanus, in which all the muscles are in a simultaneous state of contraction. In this condition the position assumed is one of equilibrium between the various muscles, and we have, in correspondence with the relative values given above, retraction of the head and trunk, the arms retracted and closely applied to the sides, the fore-arms flexed, the hands clenched and semipronated, the thighs extended, and the feet pointed and turned inwards.

So also in late rigidity the position the limb assumes is that of equilibrium between the flexors and extensors, and is not due to contracture of the flexors only, for the extensors are affected at the same time and in a similar manner.

Both relatively and absolutely the extensors of the fingers and toes are the weakest, and if we except the gluteal muscles which go with the muscles of the trunk, the extensors are relatively weaker than the flexor movements of the limbs. If we were to tax the various muscles to the same extent throughout, the extensors of the fingers would be paralysed by a tax of 5 lbs., the extensors of the wrist (cir. 7·5 lbs.

each) would be greatly enfeebled, the adductor and abductor action of the interossei similarly affected, while the muscles of the thumb, the flexors of the fingers, &c., would still retain considerable power. Similarly in the lower extremity the extensors of the toes would first be paralysed by an equal tax all round, and the peroneal muscles before the tibial.

But we are not to suppose that the depreciation of strength in the various movements, depending on any general weakening cause of the nerves and nerve centres, would be correctly represented by such a simple poll tax. The researches of Onimus clearly indicate that the vitality of the extensor nerves and muscles of the limbs becomes exhausted before that of the flexors, and apparently those of the upper extremity before those of the lower, in accordance with the order in which cadaveric rigidity attacks the various muscles.

As to what this difference depends on we can at present only speculate. But it seems not unreasonable to suppose that the degree of representation,—and therefore the trophic strength,—of the extensors in the anterior cornua is less than that of the flexors. On this hypothesis such extensors as have the most numerous connections with the spinal segments would have a greater vital resistance than those less frequently represented.

By the application of these considerations on the relative strength and vital resistance of the different nerves and muscles to the facts of saturnine paralysis, it seems to me that the hypothesis of a general peripheral neuritis, with perhaps a general impairment of the nutrition of the spinal centres, affords a more satisfactory explanation of the order of events than that of a limited anterior poliomyelitis.

Theoretically on this hypothesis the extensor communis should first be paralysed, then the extensors of the wrist, next the extensors of the thumb, then the intrinsic muscles of the hand, the supinators before the pronators; and in the upper arm the deltoid (its outer two-thirds specially) before the triceps and the flexors of the forearm. In the lower extremity the extensors of the toes, then the peroneal muscles, and in the thigh the extensor cruris would be affected before the flexors of the leg. These would be affected at a later stage than

the upper extremities, in accordance with the order of exhaustion of muscular irritability after death.

This agrees in all essential points with the usual order of invasion observed in lead paralysis. But paralysis of truly spinal origin in lead poisoning is not only possible but probable, and there is reason for believing that exposure to the influence of lead predisposes to poliomyelitis. In Case III. reported above,[1] this was a point specially noted; and in another case of poliomyelitis in a type-setter, which I have seen since, there was a strong suspicion of the action of lead. In this patient there was slight paralysis of the right deltoid, biceps, supinator longus, and extensors of the fingers, with the exception of the index finger, which acted fairly well.

Apart altogether from the powerful negative evidence as regards the spinal cord, supplied by the observations of Dreschfeld and Friedländer,[2] there are other serious objections to the hypothesis of Erb, Remak, de Watteville, &c., that lead paralysis is due to circumscribed anterior poliomyelitis. The facts of poliomyelitis in man harmonise, as I have endeavoured to show, with the types founded on experimental data, both as regards the grouping of the muscles and the relative degree in which they are implicated. But lead paralysis rarely if ever shows such correspondence. A central lesion, such as would be necessary to cause such complete paralysis of the extensors as we see in saturnine drop-wrist, would necessarily be accompanied by paralysis of the deltoid, biceps, and supinator longus, and to some extent also of the flexors of the fingers, in accordance with the facts relating to the fourth, fifth and eighth cervical types.

But, as is well known, the exception of the supinator longus in lead paralysis is almost looked upon as an axiom, though this is only true of certain stages.

And further, when the deltoid becomes severely affected, as it frequently does in saturnine paralysis, the biceps, brachialis anticus, and supinator longus may show little or no sign of impairment; a condition which does not correspond with the ordinary facts of anterior poliomyelitis.

For these reasons alone I should regard the poliomyelitic origin of lead paralysis in its ordinary form as extremely im-

[1] No. xiv. p. 236. [2] Supra cit.

probable. But when the other facts in reference to the symptomatology of saturnine affections are taken into account—the neuralgic pains, the irregular anæsthesiæ, the facts indicative of the local action on the parts most in contact with the poison; also the morbid changes demonstrated in the intramuscular nerves and nerve trunks, and the indications of a general action of lead on the muscular system,—the evidence is against limited poliomyelitis, and, while not excluding a general action on the nerve centres, cerebral and spinal, in favour of a general peripheral neuritis.

The following cases of saturnine paralysis, which I have lately had under my care, present some peculiarities, and illustrate some of the points alluded to above:—

Case V.—Charles B., ætat. 32, a plumber, which occupation he had followed from boyhood. Never had lead colic. Three or four weeks before Christmas, 1880, he complained of pain in the shoulders, and weakness of the arms, supposed to be rheumatic.

Came under my care as an out-patient at the National Hospital for Epilepsy and Paralysis on Feb. 4, 1881. The patient is thin, and the muscular system meagre. There is a well-marked blue line on the gums.

Cannot abduct his arms except to a very slight angle. Adduction is also feeble. Flexion and extension of the forearms feeble; scarcely any perceptible power of inward or outward rotation of the arms. There is well-marked drop-wrist on both sides, but more distinct on the right than left.

Grip feeble on both sides—right = 49 lbs., left = 36 lbs. The power of extension of the wrist very feeble, slightly better on the left than right. There is also faint power of extension of the fore and little finger on both sides, not of the ring or middle finger.

Electrical exploration of the various muscles by Dr. Beevor on February 14 showed that all the muscles reacted to the faradic current within normal limits, except the deltoids, rhomboids and infraspinati, and extensors of the fingers and wrist on both sides.

The contractility was abolished in the right deltoid, except in the clavicular portion, where only feeble contraction was

obtained by strong currents. The galvanic excitability was also diminished, sluggish, and anodal closure greater than cathodal closure contraction.

The contractility of the left deltoid was also greatly diminished, and the reaction of degeneration was also present.

The faradic contractility of the right infraspinatus was abolished, and to the galvanic current A C C > C C C; the left was much diminished, and A C C = C C C.

The rhomboids were also diminished, the right more than the left.

The extensors contracted to strong faradic currents on both sides, the left better than the right.

Under treatment with iodide of potassium and the constant current, progressive improvement occurred, and on August 2 the patient was practically well. He was able to raise both arms perpendicularly, extend the wrists and fingers, the latter, however, not completely. Especially was this the case with the middle and ring finger of the right hand.

The grasp of the right hand = 85 lbs.; left = 62 lbs.

All the muscles contracted readily and normally to faradic stimulation at this date.

Remarks.—This case is an illustration of the association, not uncommon, of paralysis of the extensors and deltoid, the deltoids, however, being affected relatively more than the extensors. With the deltoids also the synergic rhomboids and outward rotators of the humerus are affected.

But though the movements of both arms are abnormally weak throughout, the flexors of the forearm, viz. biceps, brachialis anticus, and supinator longus, showed no special signs of degeneration.

Among the extensors of the fingers the extensor indicis and minimi digiti was less paralysed than the others—a fact probably to be accounted for by the special representation of these actions by distinct muscles.

Case VI.—Garford L., ætat. 24, had been employed for four years at works for the extraction of silver from lead. About four weeks before coming to King's College Hospital (on Feb. 24, 1881) he had been complaining of pain and weakness in his arms, thought to be of a rheumatic nature.

On examination, the characteristic blue line at the edge of the gums was very distinct. All the movements of both arms ana hands were abnormally weak. Marked drop-wrist and total inability to extend the fingers existed on the right side. This was less visible on the left, but extension of the fingers was impossible, except feeble action of the extensor indicis and minimi digiti.

The action of the deltoids is very weak on both sides, especially on the right. He cannot put his right hand into his coat-pocket. The spine of the scapula is much more distinct on the right than left.

The following are the measurements of the two arms—the patient being a right-handed man:—

Round the axilla and acromion process .	R. = 13 in. ..	L. = 13½ in.
Round the middle of the upper arm . .	R. = 9 in. ..	L. = 9 in.
Round the thickest part of forearm. . .	R. = 9 in. ..	L. = 9⅛ in.

Electrical reactions.—Faradic contractility abolished in the right deltoid in all its parts. Reaction of degeneration to the galvanic current.

In the left deltoid faradic contractility also abolished, except slight action in the clavicular portion. Reaction of degeneration also present.

To the strongest faradic current only the extensor carpi ulnaris on the right side contracts, among the extensor group. On the left there is contraction of the extensor carpi ulnaris and extensor indicis. To the galvanic current, C C C is slightly greater than A C C on both sides.

The right supinator longus contracts to faradic stimulation, but less than the left. Galvanic reaction, A C C = C C C.

The biceps, flexors of the fingers and wrist, interossei, act well on both sides, perhaps less on the right than left.

Remarks.—In this case also the deltoids and extensors were specially affected—the deltoids relatively more than the extensors. The supinator longus on the right side, though not paralysed, yet exhibited distinct signs of degeneration.

Among the extensors, the extensor carpi ulnaris seems to have greater resisting power than the radial extensors, and the extensor indicis more than the other extensors of the fingers. Though perhaps the other muscles of the right side did not

react quite as sensitively as those on the left, yet there was no question of special impairment in any one of them.

Case VII.—William B., ætat. 30, a fishmonger, came under my care at King's College Hospital on May 7, 1881.

The present illness dates back about eleven weeks. Before this, he had had an attack of pain in the abdomen, and constipation. He cannot, however, trace any connection with lead. Lead counters were suggested as a possible cause, but he said he did not use these. There was, however, a suspicious bluish tinge of the margins of the gums.

The patient exhibited well-marked drop-wrist on both sides, and the ball of the right thumb was distinctly smaller and more flabby than that of the left. The movements of both arms were weak generally, but especially the abduction of the left arm.

Electrical reaction.—The faradic contractility of the right deltoid was fairly normal in all its parts.

In the left deltoid the faradic contractility was abolished in the scapular and acromial division, and somewhat diminished in the clavicular portion.

The left supinator longus was also somewhat diminished as compared with the right.

The contractility of the extensors of the wrist and fingers was entirely abolished, except in the extensor ossis metacarpi, and primi internodii on both sides, the right being much weaker than the left.

The contractility of the muscles of the ball of the thumb and first interosseus was almost abolished on the right, and below normal on the left.

The other interossei contract well, as also the other muscles of the arms.

Remarks.—In this case we have the association of drop-wrist on the one side with affection of the thenar muscles, and on the other with affection of the deltoid, and slightly of the supinator longus. It has been questioned whether the intrinsic muscles of the hand or the deltoids are the next in order to the drop-wrist in saturnine paralysis. Here we have a proof that

no absolute rule can be laid down, as the order was different in each arm in the same individual.

Case VIII.—John B., ætat. 30, a blacksmith by trade, came under my care at King's College Hospital on July 14, 1881.

About seven weeks ago he began to work at a furnace for the reduction of white-lead refuse, &c., to the metallic state. He enjoyed good health, and never suffered from colic.

On June 29 he found on getting up in the morning that he had great difficulty in dressing himself, on account of powerlessness in his hands. He had had some aching in his wrists, the right especially, the day before.

The patient has an earthy complexion, and the margins of the gums have a well-marked blue tint.

There is drop-wrist on both sides, specially on the right. He cannot extend the right wrist or any of the fingers, but he can extend the metacarpal bone of the thumb a little.

On the left the paralysis of the wrist and fingers is not so absolute, except as regards the middle and ring fingers. The extensors of the thumb are all retained.

The patient is very deficient in muscular power generally. The grip of the hand is weak, as also flexion and extension of the forearm, and the various movements of the upper arm. He is weaker as regards flexion of the thigh, extension of the leg, and other movements of the lower extremity, than a normal individual of the same muscular build.

Electrical reactions.—The faradic contractility is entirely abolished in the right extensor communis. Slight contraction to strong currents in the extensor ossis metacarpi pollicis.

On the left, the extensors of the thumb act feebly, and also the extensor indicis and minimi digiti to the strongest current.

The reactions of the right arm are throughout somewhat less than on the left, particularly in the scapular and acromial divisions of the deltoid, and supinator longus.

In these muscles the galvanic excitability is also diminished, but C C C $>$ A C C.

Remarks.—In this case, in addition to the greater affection of the extensors on the right side, there was also general impairment of contractility, most marked in the deltoid and

supinator longus, but visible also in the flexors of the forearm, flexors of fingers and wrist, and in the interossei.

Case IX.—Henry W., ætat. 62, a cooper, engaged principally in cleaning casks which had contained white lead, admitted into the National Hospital, Queen Square, on June 24, 1881.

The patient four years ago had an attack of paralysis on the left side.

The present illness came on three years ago, with drop-wrist on the right side. This got well, but returned again last Christmas, and he was obliged to leave off work.

The patient has a well-marked blue line on his gums.

He has a systolic murmur at the apex.

Right Arm.—Well-marked drop-wrist, and paralysis also of the extensors of thumb. Pronation and supination weak. Can flex and extend the forearm forcibly, and raise right hand to the top of the head.

Left Arm.—Drop-wrist, but not so marked as in right. Can extend the metacarpal bone of thumb, but not the phalanges. Has free movement of elbow and shoulder, and can raise the arm perpendicularly upwards, but volitional movements are all accompanied by the oscillations characteristic of disseminated sclerosis. These irregular movements have existed since the paralytic attack four years ago.

The lower limbs are moved freely, but there is a little unsteadiness in the left leg when he tries to touch a given point with the foot. The knee reaction is distinct on both sides, the left perhaps somewhat more distinct than the right. Sensibility is unaffected.

Electrical reactions.—Electrical exploration by Dr. Beevor, and subsequently by myself, showed that the faradic contractility was diminished by one half in the right deltoid, serratus magnus, supinator longus; also in the flexors of the wrist and fingers, and interossei, as compared with the left; while the biceps and triceps reacted equally and normally on both sides.

The contractility was entirely abolished in the extensors of the fingers and wrist on both sides.

In the right, the extensors of the thumb showed no reaction;

while on the left, the extensor ossis metacarpi pollicis contracted to moderate currents. The galvanic excitability of the extensors of fingers and wrist was also abolished.

Remarks.—In this case, of long standing, the degeneration of the extensors was so complete as to have resulted in entire abolition of faradic and galvanic excitability, while the other muscles of the arms enjoyed practical immunity. But that they had suffered in the right arm is shown by the diminution of excitability generally, and specially in the deltoid and supinator longus. The flexors of the forearm,—biceps and brachialis anticus, had not suffered appreciably.

This case is another illustration of the fact exemplified also in Cases 6, 7, 8, that the supinator longus does not always escape in saturnine paralysis, and is liable to degeneration along with the deltoid in the severer forms.

I would conclude this brief survey of the facts relating to the localisation of atrophic paralyses with a few remarks on *Progressive Muscular Atrophy.*

Though the primary spinal origin of progressive muscular atrophy is generally accepted, yet as actual morbid changes in the anterior cornua have not been demonstrated in all cases of this nature, there may appear, taking these facts alone, some grounds for the theory maintained by Friedereich, and more recently again advocated by Lichtheim [1] that muscular atrophy is primarily a myositis. But in addition to the arguments founded on the analogy of bulbar paralysis, and on the deuteropathic muscular atrophy which is not unfrequently associated with lateral sclerosis, sclerosis of the posterior columns, syringo-myelitis, spinal injuries, &c., the order in which the various muscles of the limbs are invaded by the atrophic degeneration, and their mode of grouping, afford one of the strongest arguments in favour of the spinal origin of protopathic muscular atrophy.

The favourite commencement of muscular atrophy is in the intrinsic muscles of the hand, according to most observations in the ball of the thumb and first interossei. From the hand

[1] 'Archiv f. Psychiatrie,' viii.

it spreads to the muscles of the forearm, specially affecting the flexors and pronators, and relatively sparing the extensors and supinators. The usual result is to develop a characteristic "main à griffe," which depends on the unantagonised action of the extensors of the basal phalanges consequent on the disappearance of the interossei. This mode of advance is precisely in accordance with progressive invasion of the spinal segments corresponding to the first dorsal and eighth cervical roots. Though the extensors, and particularly the extensor ossis metacarpi pollicis, are more or less implicated, yet, even when the atrophy has advanced to almost complete disappearance of the intrinsic muscles of the hand and long flexors, the extension of the wrist and fingers may be carried out with considerable force. Though, as has been stated, the extensors, as being synergic with the intrinsic muscles and long flexors, are represented in the eighth cervical segment, yet, as they are again represented in the fourth and fifth cervical segments, they will naturally retain considerable power so long as these segments are intact. But when these are invaded, entailing atrophy also in the deltoid and other muscles of the upper arm, the extensors also waste, and the characteristic "griffe" does not occur. The escape of the supinator longus, relatively more striking than that of the extensor group, is also readily explicable by its representation along with the flexors of the forearm in the upper two cervical segments. The supinator longus should, according to these data, go with the flexors of the forearm, deltoid and other muscles of the upper arm, and of this association there are many examples already on record. It has been remarked by Duchenne that the triceps is the last muscle to be affected by muscular atrophy. This is also in accordance with the position and representation of this muscle in the various segments of the cervical enlargement. Being represented in the eighth, seventh, and sixth cervical roots, and more frequently than any of the other muscles which have their centres here, it is natural that it should retain its functional activity and nutrition in some degree, till the whole of this region has become invaded. And as it seems to be the rule for the degeneration to proceed either from the lower or upper end of the cervical enlargement, or it may be from both

simultaneously, the centres for the triceps will naturally be long in being completely involved.

The order in which the muscles are successively attacked by progressive atrophy is thoroughly in harmony with the mode of collocation and order in which they are represented in the spinal segments, and with the advance of a degenerative process in the multipolar cells of the anterior cornua from below upwards or above downwards; whereas it is altogether impossible to reconcile the facts with Friedereich's hypothesis of a primary myositis spreading merely *per contiguitatem* and limited by the large joints. This is unable to explain the association of the supinator longus with the flexors of the forearm, or the spread of atrophy from the intrinsic muscles of the hand, more particularly to the long flexors, and many similar facts. Friedereich's view, that the changes in the anterior cornua, which have been found in a certain number of cases, are merely secondary to the degeneration primarily originating in the muscles, so far finds support in the apparently demonstrated absence of morbid changes in the spinal cord in other cases. But we have good reasons for believing that, in future, cases of muscular atrophy without changes in the anterior horns will be less frequently recorded than heretofore, if the various segments of the spinal cord are investigated with the requisite degree of care and minuteness. It is not improbable that the extreme upper and lower extremity of the cervical enlargement may sometimes have escaped investigation. But it will be apparent from what has preceded, that the segments corresponding to the first dorsal and fourth cervical roots are of all others the parts demanding special investigation in atrophic paralysis, commencing with the hand or upper arm respectively.

THE PHENOMENA OF CONVULSION.

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THE storage and expenditure of force which it is the province of the nerve cells to effect are, and must ever be, hidden from direct observation and measurement. Their effects are displayed in muscular contraction only, and the whole science of Neurology is based on the assumption that the extent and the manner of muscular contraction correspond with and represent the extent and the manner of the nervous discharge. How far and under what restrictions this assumption is true, it will be the object of a subsequent paper to examine; but, accepting it provisionally, it is obvious that to gain a clear insight into the processes going on during morbid action of the nervous centres, it is necessary to observe with the greatest care the phenomena of morbid muscular contraction.

The phenomena of convulsive seizures have amid all their variations so fundamental a similarity among themselves, in comparison with their wide differences from the other morbid kineses of the human body, that they form an extremely well-characterised group of phenomena, and to be studied advantageously must be studied together.

The time has gone by when proof would have been required of the fundamental identity of the process occurring in all convulsive seizures, whether of "idiopathic epilepsy," uræmia, hydræmia, cerebral tumour, or accompanying other pathological conditions. However necessary it may be for the clinical physician to regard separately the convulsions occurring in different pathological states, or to look on "idiopathic epilepsy" as a clinical entity, the pathologist must recognise that the nosological position of epilepsy is much the same as that

of dropsy, in other words, that it is indeed a symptom which may be associated with, and even dependent on various pathological conditions.

As the essential nature of dropsy is an excess of transudation into the tissues over absorption from them, so the essential nature of epilepsy is an excess of tension of the molecular force stored up in the grey matter over the resistance to its discharge. As the disturbance of the balance between transudation and absorption, which constitutes dropsy, may depend on very various pathological conditions, so the upsetting of the equilibrium between tension and resistance in the grey matter, which constitutes epilepsy, may depend on very various pathological conditions.

That there are various forms of convulsion, and that these forms are divided by profound differences is unquestionable; but these differences must be studied apart from their clinical and pathological concomitants, with which they have little or no correspondence.

Regarded from this point of view, the factors which go to make up the *ensemble* of a convulsion are ten, viz.:—(1) the Quality, (2) the Origin, (3) the March, which, together determine (4) the Attitude assumed by the convulsed part, (5) the Extent, (6) the Rapidity, (7) the Pitch, (8) the Intensity, (9) the Force, and (10) the Duration.

The most fundamental distinction between the different forms of convulsion is the quality of the spasm, which may be either tonic or clonic. By clonic spasm is here meant a spasm during any part of which the simple contractions that compose it are separately perceptible. The characteristic of clonic spasm is therefore its intermittent character. It is true that at the height of the fit the simple contractions succeed each other so rapidly that the spasm is continuous, but it is so at the height of the fit only, and, as the severity of the seizure declines, the simple contractions are severed by a greater interval, they become discrete, and separately perceptible.

By tonic spasm is meant a spasm in which the separate simple contractions never become perceptible. Its characteristic is its continuous nature. Conformably with this, it is found to remain present for many hours and days together;

undergoing, indeed, exacerbations and remissions, which are always, however, of the same quasi-continuous character, having the form of waves of deliberate rise, progress and decline, and which exhibit a marked absence of suddenness. The chattering of the teeth may be taken as a normal example of the clonic, and a yawn as a normal example of the tonic form of spasm.

It will be observed that the terms "tonic" and "clonic" are here used in a somewhat different sense from that usually attached to them.

The continuous or intermittent quality of the spasm, while of itself it impresses a marked individuality on each variety of convulsion, is the index to so many and such wide other differences as completely to justify its selection as the basis of classification.

For the convenience of further description it will be necessary to consider the muscles of the body as divided into series, each series having its base at the trunk, and its apex at the periphery. Thus one series passes through buttock, thigh, leg and foot to the toes, the muscles of which form the highest order of this series. Another series on each side passes through shoulder, arm, forearm and hand to the fingers, the muscles of which form the highest order of this series. Again, another series passes up each side of the neck, and, bifurcating, has two topmost orders, at the eye and at the mouth, the former being the higher. The muscles of each of these series must be considered as arranged in orders, which correspond roughly with the sizes of the muscles, and more nearly with their position in the series; the muscles of the lowest orders being the largest, the more often bilaterally acting, those having the most general functions, attached to parts having the fewest, samest and least precise movements, and whose action is attended with the least amount of conscious voluntary effort. Such muscles are the diaphragm, the muscles of the back, and of the jaws. At the other end of the scale are those muscles having the most special functions, attached to parts having the most numerous, most varied and most precise movements, acting unilaterally and with the greatest amount of conscious voluntary effort. Such muscles

are those of the eyes, and those of the thumb and fingers. On this scale the muscles which close the fist will be of higher order than those which move the fingers in writing, and those which move the wrist, elbow and shoulder, will be of successively lower and lower orders.

The origin of a convulsion referred to this scheme, is always at the apex or the base of a series, never in the middle.

The importance of attending to the point of origin of a convulsion has long been insisted on by Dr. Hughlings-Jackson, who also pointed out that unilateral convulsions begin in those muscles having the qualities above enumerated, and which are here called of the highest order.

The commonest opening of a convulsion is the deviation of the eyes to one side. Next in order of frequency comes spasm of the thumb and finger. Spasm of the lips, although it nearly always follows deviation of the eyes, is far less common as an opening, and convulsion beginning in the foot is comparatively rare.

It is less usual for a fit to begin in the lowest orders—the muscles of the back or of the jaws—but the fits of so-called "idiopathic epilepsy" do occasionally begin in this region, and the spasms of tetanus and of strychnia-poisoning always begin in, and are generally confined to these, the most general of all the voluntary muscles. Lastly, a convulsion may begin in the most general of all muscles, those of the blood-vessels and of the skin; as in the case of a rigor, which is an epilepsy of the vaso-motor and its allies.

It may seem unwarrantably foolhardy to suggest that phenomena so diverse as an epileptic fit, a tetanic spasm and a rigor should be studied together, and to do so for clinical purposes would be truly preposterous, but the contention is that they are all instances of sudden excessive and transient muscular spasm, due to some process taking place in the nerve-centres, and that in order to investigate this process it is necessary to dissociate from their surroundings all cases in which it occurs and to study the cases together. In other words, however far apart they may be clinically, yet pathologically an epileptic fit and a rigor are closely allied, and it is with the pathological aspect of convulsions that this paper has to do.

To say that an epileptic fit and a rigor are pathologically allied is not a mere assumption, for it is well known that the specific fevers, which in the adult are ushered in by a rigor, are in children frequently heralded by a convulsion which replaces the rigor.

It will depend of course on the origin of the spasm whether the March is centripetal or centrifugal; but whether the fit begins in the highest order and spread downwards (centripetal), or whether it begins in the lowest order and spread upwards (centrifugal), the march or order of spreading is threefold. Only in the former kind, however, can this triple order be clearly distinguished.

The three directions in which simultaneously a centripetal convulsion spreads are :—first, the muscles first involved become more severely affected: second, the spasm affects additional muscles of the same order as those first affected: third, it spreads to the muscles of the order next below in the same series. The further spread of the convulsion follows precisely the same method, with this addition, that when it has reached a certain depth in the series in which it began, it begins to affect another series on the same side of the body. This second series is invaded generally from the lowest order to the highest, and sometimes probably from the highest order to the lowest, but very often all its orders are involved apparently simultaneously. It is most probable that there is always a definite march; but fits that spread as far as this are usually of rapid progress, and by the time the spasm has spread thus far, it has become so highly compound, and the appearances are so fleeting, that observation has become extremely difficult.

As the fit progresses, another and another series is affected by the convulsion, until the whole body is convulsed. The order in which the series are taken varies. The limb transversely opposite to that first affected is always affected centrifugally when any march can be discerned. The limb diagonally opposite to that in which the fit began is affected last and least.

The cessation of the spasm is usually simultaneous, or nearly so, over the whole body. Occasionally the part first affected can be seen to be last released.

The above description is necessarily complicated, because the phenomena it describes are complex. An instance may render the matter clearer. Suppose a convulsion begins in the thumb and finger. The first step in the march will consist of three elements, thus: (1) the spasm in the thumb and finger becomes more severe, (2) the muscles of the other fingers (more muscles of the same order) become affected; and (3) some of the muscles moving the wrist (muscles of the next order) are slightly affected. The next step will similarly consist of three elements: (1) the spasm of all these parts becomes more severe; (2) more of the muscles moving the wrist become implicated; and (3) some of the muscles moving the elbow are slightly affected. The third step will be essentially similar, in so far as the arm is concerned, but in addition to this, the head and eyes begin to turn to the affected side, showing the implication of an altogether new series of muscles. As the spasm of the arm becomes more severe, so does that of the face and neck, and in addition the arm of the opposite side and the leg of the same side become involved; the leg of the opposite side being the last to join.

The merit of directing attention to the origin and march of convulsions belongs entirely to Dr. Hughlings-Jackson, by whom also the order of march above described was discovered. It has been verified by the writer in very numerous observations.

In a subsequent paper it will be shown that the three elements in the march of a convulsion correspond with the spread of the discharge in the nerve-centres in intension, in extension, and in depth respectively.

There is yet another way in which a convulsion may spread. Without increase of depth, i.e. without involving fresh orders of muscles, it may affect successively different combinations of muscles of the same order, causing the part affected to assume successively different attitudes. For instance, supposing the thumb and two adjoining fingers to be at first extended and the other fingers flexed, then, as the fit continues, the thumb may be flexed and the ulnar fingers extended, without the spasm existing elsewhere undergoing any material alteration. The process in the nerve-centres which this mode of spread-

ing represents is a shifting of the dominant focus of the discharge.

Centrifugal convulsions, beginning in the lowest order of muscles, that which forms the point of meeting of all the different series, affect, at the first step of their march, the next lowest orders of all the series simultaneously. That is to say, beginning in the muscles of the back, the spasm spreads simultaneously to the thighs, the upper arms and the jaws.

The attitude assumed by the part under the influence of the spasm bears a definite relation to the quality, origin, and march of the convulsion. It may be stated generally that

All tonic convulsions are bilateral, and produce an attitude of extension.

All centrifugal convulsions are bilateral, and produce an attitude of extension.

The remaining class of convulsions, comprising all those which are clonic in quality and of centripetal march, begin unilaterally and produce an attitude of flexion.

Tonic centrifugal convulsions are very rarely complete, rarely, that is, affect the whole body; when they do so, the attitude is as follows. The head is thrown back, the trunk is arched backwards, the jaws may be open or closed, but, if affected at all, are of course immobile. The arms are extended by the sides, the elbows extended to the utmost, the forearms pronated, the hands in the "tetany position." The thighs, knees and ankles are extended, and the toes flexed. The eyes are affected only in the most extreme cases, and are then rolled upward. To this class belong the convulsions of tetanus, of strychnia-poisoning, and of cerebro-spinal meningitis.

Tonic centripetal convulsions affect both hands simultaneously or nearly so, and when the spasm reaches the shoulders the feet become affected. It does not often extend far enough to retract the head, but, were it ever to become complete, would probably produce an attitude the counterpart of the last. The fingers are extended, except at the knuckles, which are flexed, the thumb lies against the second internode of the index, the elbow is extended, and the forearm in severe cases pronated. The feet are strongly extended, everted, and

the toes flexed. The flexion of the knuckles is an apparent, not a real exception to the prevailing extended attitude, for it is part of the "interosseous position," and the interossei are primarily extensors of the distal joints of the fingers. The flexion of the knuckles is quasi-accidental, and subordinate to the extension of the other joints. Tonic centripetal convulsions are exemplified in tetany.

Clonic centrifugal convulsions are in many respects intermediate in character between the bilateral extending and the unilateral flexing convulsions. The spasm is of a more uniform character than that of typical clonic convulsions, its accession and decline are more gradual; the attitude often appears a compromise between flexion and extension, the former preponderating in the legs and the latter in the arms and hands; the duration of the fit, too, is longer and the intensity less than is usual in clonic convulsions. The general attitude is still one of extension, and simulates the type described above, the more closely according as the march is more definitely centrifugal. This form of convulsion is seen in certain cases of "idiopathic epilepsy."

Clonic centripetal convulsions, which form by far the largest and most varied group of these phenomena, and include nearly the whole of the so-called epileptic, epileptoid and epileptiform convulsions, invariably produce an attitude of flexion. The first is closed, the thumb bent into the palm, the wrist and elbow bent, the shoulders brought forward, the head bowed, the eyes drawn to one side, the eyelids closed, that one on the side to which the eyes are drawn more tightly than the other, the lips and tongue also are drawn to that side. The spine is flexed, the legs drawn up, the feet inverted and flexed. This form of convulsion always begins in the highest order of a single series on one side, and affects that series and that side most.

The attitude assumed by a convulsed part depends on the position of the dominant focus of the discharge. By the extent of a convulsion is not here meant the area of the body or the number of muscles involved in it, but the degree to which its threefold march as above described has extended; and it is measured by the *order* of muscles which the spasm

has reached. The varying extent of the spasm is one of the main causes of the different aspects that convulsions present. A convulsion may be limited in extent to a mere quivering of one eyelid—"live blood," as it is termed; or it may add to this a retraction of that side of the mouth and a deviation of the eye; or it may superadd a turning of the head and drawing up of the arm; or it may extend to universal convulsion; but between these various degrees no sharp line of demarcation can be drawn, they are but variations of the same phenomenon, extensions of the same process, and their fundamental identity is shown by the fact that the same patient may at short intervals of time exhibit convulsions varying in extent as widely as those instanced above, yet all having the same origin and spreading in the same way. From the above description of the threefold march, it will be seen that the greater extent of the fit, the more severely are those parts affected that were first seized.

By the rapidity of a convulsion is meant that of its progress from part to part of the body, or from order to order of muscles. The rapidity is always considerable, and often so great that the convulsion appears to affect the whole body suddenly and simultaneously, and from this cause arises mainly the difficulty, often extreme, of accurately observing convulsions.

In fits of deliberate march the spasm may be three or four seconds in spreading from muscles of the highest order to those of the lowest, and seven or eight in becoming universal. It is very rarely slower than this. In rapid fits, in which the convulsion appears to be universal from the very outset, it may be impossible to determine the point of origin, except inferentially by observing the part *most* affected. Between these extremes there is every degree of rapidity. Fits of deliberate march are usually due to gross disease of brain-tissue, and it may even be said with some approach to accuracy that the coarser the structural alteration, the more deliberate the march. Thus the convulsions whose outset is slowest of all are those arising from tumour of the brain; more rapid are those of meningitis, while the fits of "idiopathic epilepsy," in which the change in brain-tissue is so fine as to be unrecognisable, are extremely rapid. This fact is not unin-

teresting from a pathological point of view, for a tumour may be regarded as a local change in an otherwise normal brain which overcomes with difficulty the stability of the normal elements; while in "idiopathic epilepsy" the whole grey matter may be regarded as in an habitually unstable condition, so that a disturbance once begun will travel rapidly. It is the difference between the spread of a fire through an ordinary dwelling-house, and that through a bare house filled with combustibles. In conformity with this view, it is found that when convulsions have occurred repeatedly, from whatever cause, the latter attacks spread more rapidly than the earlier ones, as if the more frequent discharges had made the elements permanently more unstable.

There is another element in the rapidity of a convulsion irrespective of its march from muscle to muscle. The continuous contraction of a muscle is the product of a great number of contractions rapidly succeeding one another. Each of these "simple contractions" is due to a separate delivery of force from the nerve-centre through the nerves into the muscles. In the convulsive contraction of a voluntary muscle the same condition of course obtains; but the simple contractions are of a different strength, and follow one another at different intervals from those which compose a normal contraction. The rapidity with which their shocks of simple contraction follow one another I propose to call the "Pitch" of the spasm, borrowing a term from the obvious analogy between the composition of a muscular contraction and that of a musical note.

The pitch of the spasm, as thus defined, varies greatly both in different fits and in different periods in the same fit. The pitch bears some relation to the rapidity of spread, that being higher as this is more rapid, but the two do not maintain a very constant ratio. In any one clonic convulsion the pitch is always high at the commencement, and is highest either at or very shortly after the opening. Towards the end the pitch *always* declines, its declension proceeding *pari passu* with that of the intensity of the fit, of which it forms a main element. The first indication of the lowering of pitch is that the muscles, hitherto contracted into a rigid fixed mass, begin

to quiver, the less rapid succession of shocks allowing of a slight relaxation of the muscle between their arrivals. As the pitch runs down, the quivering widens into a succession of twitches, at first slight, others becoming more and more marked as the longer intervals allow the relaxation between the successive shocks to become more and more complete. At the end of the fit the pitch reaches its lowest point, the intervals between the shocks becoming so prolonged as to allow of complete relaxation of the muscles, and, after two or three entirely separate and discrete "simple contractions," the convulsion ceases.

The pitch is not always uniform over the whole body, but often appears to be less uniform than it really is. For if the successive shocks of simple contraction are but slight, they may suffice to give perceptible movement to the more mobile parts, e.g. the lips and eyelids, but may fail to set in motion heavier parts such as the arms; although by grasping the arm its muscles may be felt to be affected by a feeble but sudden hardening, synchronous with each contraction in the face.

Fits of long duration soon become less highly pitched, and in very prolonged convulsions the simple contractions may be separated by intervals of one or more seconds. In these prolonged convulsions the pitch of the spasm is seen to have a compound rhythm. Among the twitches due to single contractions may be observed displacements of a more gradual character, which are due, as may be ascertained by grasping the muscles, to waves of spasm each containing three or four or more simple contractions. And at longer intervals occur still greater contortions caused by the fusion of several such waves. From time to time the contortion becomes so great, and so prolonged as to deserve the name of a separate fit, and from the way in which they occasionally merge into one another, we are justified in regarding the phenomena of the prolonged convulsion as representing those of the rapid convulsion "magnified in time." Hence the observation of this somewhat uncommon form is of extreme value. It may here be remarked, in parenthesis, that all forms of tremor are due to lowering of the pitch of contraction. I believe that the height of the pitch depends on

the proportion which the number of active elements bears to the number of active centres.

The "simple contractions" that go to make up a muscular spasm vary not only in the rapidity with which they succeed each other, but also in intensity, by which is meant the extent to which each contraction shortens or tends to shorten the muscle. Supposing the succession of simple contractions to be represented graphically by a series of waves, then would the pitch be represented by the length, and the intensity by the amplitude of the waves. In respect to the total spasm, the degree of intensity determines the amount of contortion, or the difference between the attitude assumed by the convulsed part and its attitude when at rest.

The intensity of the spasm increases continuously from the opening of the fit until it reaches a maximum, and remains at this point to the very end of the fit. Remembering that the contractions occur at longer and longer intervals towards the end of the fit, while their intensity and their suddenness remain unaltered, it will be seen that as the muscles relax more and more between the contractions, the amount of actual movement produced by them is increased, and thus a fit often appears more severe, when it is in reality passing off.

The pitch and the intensity together make up the force of the contraction, which is measured by the amount of resistance that it opposes to alteration of the attitude of the convulsed part by passive movement. It is extremely probable that there is another element in the composition of the force of the spasm, viz.—the force of the individual simple contractions apart from their amplitude, an element which would be represented in the graphic scheme above referred to by the *width* of the undulations. But beyond *à priori* probability, no evidence of the existence of this element is at present forthcoming.

Lastly, the duration of a convulsion may be so transient that a group of observers standing before the patient may be divided in opinion as to whether one has taken place at all, or it may be prolonged for hours and days. The longest continuous convulsion that has come under the notice of the writer, lasted for ninety-six hours.

The ten elements above enumerated make up the whole of the phenomena of convulsion proper, and the various forms of convulsion are due to their combinations in varying proportions. Regard being had to the number of the elements and the wideness of the limits within which they may vary, the assertion will be seen to be justified that a rigor and an epileptic fit, the twitching of an eyelid and opisthotonic spasm, are phenomena of identical nature, due to similar nervous processes, which, however, are of differing extents and affect different nervous centres.

And since, to return to the initial proposition, every one of these elements must correspond with and be a measure of some *modus* of the process (discharge) in the nerve-centres which gives rise to the convulsion, it is obvious that to obtain an insight into the nature and *modus* of this process, it is necessary to study and record the various elements of the convulsion, their degrees and their ratios to one another. To advocate such study is the object of this paper.

It is scarcely necessary to remark on the difficulty of observing so many component threads, woven into such a complicated texture, and passing before the eye with such rapidity as the phenomena of a clonic convulsion. The difficulty is always great, often extreme, and is surmountable only by watching a great number of fits, and in each one singling out some one element for observation. No doubt the method is faulty, but it is the best we have. No two fits are ever quite alike even in the same patient on the same day, but, on the other hand, all the fits of any one patient show a very great approach to uniformity in their Quality, Origin, and March, and differ only in the minor elements; and hence the observation of successive fits in the same patient affords the most valuable information as to the influence of these minor elements on the concomitants and sequelæ of the fits.

The occurrence of these, the secondary effects of the discharge, still further complicates the convulsion, and intensifies the difficulties of observation. For example: it is impossible to tell how far the quickening of the heart's action and the lowered tone of the arteries are due directly to the action of the discharge, how far they are due to it indirectly by the

exhaustion which succeeds it, and how far doubly indirectly by the interference with respiration.

All the indirect effects of the discharge, concomitants and sequelæ of the convulsion, comprising the mental phenomena, auræ, &c., the loss of consciousness, paralysis, automatic actions, vascular and glandular disturbances, have been purposely omitted from consideration, and the attempt has been made to treat of convulsions purely in terms of muscular contraction.

It will be the object of a subsequent paper to elucidate the various modes of the nervous discharge with which these elements correspond.

EPIDEMIC CONVULSIONS.[1]

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EXTRAORDINARY interest was excited in the popular mind of Kentucky, at an early day, by a form of convulsive disease, which, though it had been witnessed elsewhere in the world, had never before assumed a shape so decidedly epidemic. Among the Camisards or French Prophets, who appeared in the mountains of the Cevennes toward the close of the 17th century, the subjects, when about to receive the gift of prophecy, were often affected with trembling and fell down in swoons. When the fit came, no matter where they were, they fell, smiting their breasts with their hands, crying for mercy, and imprecating curses on the Pope. They were finally, after an obstinate struggle, put down by their insane persecutor, Louis XIV.[2]

Epidemic convulsions prevailed in Scotland, half a century later. Multitudes, under pungent preaching, were violently agitated, uttering loud cries, shaking, trembling, bleeding at the nose, the minister promoting the uproar by urging them not to stifle their convictions. The shriek, or the shout, it is stated, never rose from one, but that others joined the outcry.[3] The early career of John Wesley is well known to have been marked by similar disorders. In his journal he records numerous instances of men and women dropping to the ground

[1] The larger part of the materials contained in this paper were collected by my father, the late L. P. Yandell, M.D., and were intended to be embraced in the 'Medical History of Kentucky,' a work on which he was engaged at the time of his death. I have done little more than arrange and place them in their proper chronological order.—D. W. Y.

[2] Encyl. Amer., art. "Cevennes."

[3] Rees' Cyclopedia, art. "Imitation."

under his preaching "as if struck by lightning," ten or a dozen praying at once. They had also prevailed extensively in New Zealand half a century before they became epidemic in Kentucky. The elder Edwards has left an instructive account of the bodily agitations which accompanied the revivals of religion from 1735–1742. Many instances are given of fainting, falling, trance, numbness, outcries and convulsions, and he relates that some of the subjects lost their reason.[1] The epidemic of Kentucky spread more widely, and persisted for a longer time, as well as in more extravagant forms. It continued to reappear for several years, and involved a district of country extending from Ohio to the mountains of Tennessee, and even into the old settlements in the Carolinas. Lorenzo Dow relates that, at a religious meeting in the court-house of Knoxville, when the Governor of Tennessee was present, he saw one hundred and fifty people "jerking" at one time. But at other places the frenzy reached a greater height. It was computed that at a religious meeting in Kentucky, not less than three thousand persons fell in convulsions to the ground.

The extraordinary religious excitement in which these nervous disorders took their rise, commenced in Logan county, Kentucky, under the preaching of Rev. James McGready, described as a man of "hideous visage and thunder tones," with a highly impassioned style of eloquence.[2] The excitement abated soon, but was renewed in a more intense form three years later, and continued to grow and deepen until it reached its height about the year 1800. Its effects were described by this fiery preacher as at that time "exceeding everything his eyes had ever beheld upon earth." Families came in waggons, 40, 50, and 100 miles to attend the meetings, and it became necessary to establish camps for their accommodation. These camp meetings generally continued four days, from Friday to Tuesday morning, but sometimes they lasted a week. One succeeded another in rapid succession, and thus the fervour of religious feeling was kept up. The woods and paths leading to the camp ground seemed alive with people. "The labourer," says Dr. Davidson, in the

[1] Edwards on 'Revivals.'

[2] Dr. Davidson's 'History of the Presbyterian Church in Kentucky.'

work just quoted, "quitted his task; age snatched his crutch; youth forgot his pastimes; the plough was left in the furrow; the deer enjoyed a respite upon the mountains; business of all kinds was suspended; dwelling-houses were deserted; whole neighbourhoods were emptied; bold hunters, and sober matrons, young men, maidens, and little children, flocked to the common centre of attraction; every difficulty was surmounted, every risk ventured, to be present at the camp-meeting."

The concourse became immense. At one of these assemblages the attendance was computed at 20,000 souls. And here were united all the elements best suited to stir the emotional nature of man, and to derange his nervous system. The spectacle at night, as Dr. Davidson depicts it, was one of the wildest grandeur. With great beauty of description, he says, "the glare of the camp fires, falling on a dense assemblage of heads simultaneously bowed in prayer, and reflected back from long ranges of tents upon every side; hundreds of candles and lamps suspended among the trees, together with numerous torches flashing to and fro, throwing an uncertain light upon the tremulous foliage; the solemn chanting of hymns swelling and falling on the night wind; the impassioned exhortations, the earnest prayers, the sobs, shrieks, or shouts, bursting from persons under intense agitation of mind; the sudden spasms which seized upon scores, and unexpectedly dashed them to the ground—all conspired not only to invest the scene with terrific interest, but to work up the feelings to the highest pitch of excitement." To these circumstances, that tended so powerfully to excite the nervous centres, we have to add others which gave intensity to their effect. The meetings were protracted to a late hour in the night, keeping the feelings long upon the stretch. A reverent and general enthusiasm ascribed the bodily agitations to a mysterious, Divine agency. The preaching was fervid and impassioned in the extreme. Many of the preachers, unable to control their emotions during sermon, went around in "a singing ecstasy," shouting and shaking hands with others, as much excited as themselves. In this way everything was done to "heap fuel

[1] Dr. Davidson's 'History of the Presbyterian Church in Kentucky.'

on the fire," and it was at such meetings that thousands fell in convulsions to the ground.

Some of the actors in these strange scenes have left records of the state of their minds, which show that they were in a condition bordering on insanity, if not actually insane. One of them relates that while under conviction on account of his sins he went about the woods for two years, through rain and snow, "roaring, howling, praying, day and night." And when light and hope broke in at last upon his mind, which he describes as a "rushing, mighty wind, that descended from heaven, and filled his whole being," he went shouting over the encampment all night and a great part of the next day. He continues: "I now made the mountains, woods and canebrakes ring louder with my shouts and praises than I once did with my howling cries; I never fell on my knees in secret but the Lord poured out His power, so that I shouted out aloud. Sometimes I shouted for two or three hours, and even fainted under the hand of the Lord. I was ready to cry out at the name of Jesus. The brightness of heaven rested continually upon my soul, so that I was often prevented from sleeping, eating, reading, writing or preaching. I would sing a song, or exhort a few minutes, and the fire would break out among the people. I have spent nine nights out of ten (besides my day meetings, and long, hard rides) with the slain of the Lord."[1]

Granade is the preacher who gives this description of himself, which is also descriptive of his times. He was a stormy orator who drew great crowds wherever he went. He admits that he went by the name of "the distracted preacher," but says that at one of his meetings "the people fell as if slain by a mighty weapon, and lay in such piles and heaps that it was feared they would suffocate, and that in the woods." So violent was his manner, stamping with his feet, and smiting with his hands, that he often broke down the stands erected for him in the woods. Once, it is told of him, he was addressing a class meeting in the upper story of a dwelling-house, when the room below was crowded with worshippers, and being in what the historian calls "one of his big ways," he exclaimed, "I feel like breaking the trigger of hell," and at the same

[1] McFerrin's 'Methodism in Tennessee.'

time gave a tremendous stamp with his foot which actually broke one of the joists. The people below, hearing the sudden crash, ran screaming to the door, some of them really imagining, as the writer of all these events relates, "that hell had overtaken them."[1]

Granade was of an excitable temperament, and vivid imagination. His person was commanding, and with a sounding voice and most impassioned manner, his oratory produced startling effects.

Another feature of these excited meetings which served still further to intensify the feelings of the people who attended them for days and nights together, was the part taken in them by children. Nothing was more affecting to the congregations than the sight of a little boy or girl on a log or stump, passionately exhorting the multitude. Thus, a boy who appeared to be about 12 years of age, is described as having retired from the stand at Indian Creek, Ohio, during the sermon, and mounting a log, and raising his voice to a high pitch, soon had nearly all the congregation with him. "With tears streaming down his cheeks, he cried aloud to the wicked, warning them of their danger, denouncing their certain doom if they persisted in their sins, expressing his love for their souls, and desire that they should turn to the Lord and be saved." A man on each side held the boy up, and he spoke for about an hour. When quite exhausted, and language failed to give utterance to his emotions, the little orator raised his hands and, dropping his handkerchief wet with tears and perspiration, cried out, "Thus, O sinner, shall you drop into hell, unless you forsake your sins and turn to the Lord." At that moment, the writer of this account continues, "Some fell like those who are shot in battle, and the work spread in a manner which human language cannot describe."[2]

McNemar instances boys of 8 and 10 years, and the Rev. John Lyle mentions one of 7, who called on sinners to repent, with an eloquence singularly overpowering. Possessed by one dominant idea, the people gave themselves up to the wildest enthusiasm, and it was no uncommon thing for them

[1] McFerrin's 'Methodism in Tennessee.' [2] Ibid.

to spend the whole night in religious orgies such as have been described.[1]

The spectacle of persons falling down in a paroxysm of feeling was first exhibited at Gasper River Church, in one of McGready's congregations in the summer of 1779. The movement proved highly contagious and spread in all directions. After a rousing appeal to the feelings of the listeners, and especially during spirited singing, one and another in the audience would fall suddenly to the ground and swoon away. Not only nervous women, but robust young men were overpowered. Some, continues the historian, fell suddenly as if struck by lightning, while others were seized with a universal tremor before they fell shrieking.[2] Dr. Blythe, who often witnessed scenes of this sort, assured Dr. Davidson that he had once felt the sensation himself, and only overcome the tendency to convulsion by a determined effort of his will. A few shrieks never failed to put the assembly in motion, and set men and women to falling all around. A sense of "pins and needles" was complained of by many of the subjects, and others felt a numbness of body, and lost all volitional control of their muscles. It soon grew into a habit, and those who had once fallen were ready to fall again under circumstances by no means exciting. Women who had suffered repeated attacks sometimes fell from their horses on their way to or from the meeting-house, while relating their past religious exercises.

The condition in some of the subjects was cataleptic, lasting generally from a few minutes to two or three hours; but in a few cases it continued many days. Others were violently convulsed as in hysteria or epilepsy, "wrought hard in fitful nervous agonies, the eyes rolling wildly." Most were speechless, but some were capable of conversing throughout the paroxysm. The extremities were cold; the face was pale or flushed, the breathing hard. Sensibility was annulled. Mr. Lyle, one of the prominent preachers of the times, having been furnished by Dr. Warfield with a vial of hartshorn, applied it to a stout young man who was lying flat on his back, and, inadvertently, let some of the fluid run into his

[1] Davidson, op. cit.

[2] Lyle's 'Diary.'

nostrils; but he took not the slightest notice of it.[1] Others who fell hard to the ground, or in running encountered stumps or trees, felt no pain from the violence. So many fell at Cabin Creek camp-meeting, it is related, that to prevent their being trodden upon, "they were laid out in order on two squares of the meeting-house, covering the floor like so many corpses." At Paint Creek Sacrament 200 were estimated to have fallen; at Pleasant Point 300 were prostrated; while at Cane Ridge, as has been stated, the number who fell was believed to have reached 3000.

The "Jerks," as they were termed, presented some novel and remarkable features. Their first occurrence is reported to have been at a sacramental meeting in East Tennessee, where several hundred people of both sexes were seized with this strange, convulsive movement. The Rev. B. W. Stone has left a vivid description of it. Sometimes, he says, the subject was affected in a single member of his body, but at others the spasms were universal. When the head alone was affected it would be jerked from side to side so quickly that the features of the face could not be distinguished. When the whole system was affected, he continues, "I have seen the person stand in one place, and jerk backward and forward in quick succession, the head nearly touching the floor behind and before. All classes, saints and sinners, the strong as well as the weak, were thus affected. I have seen some wicked persons thus affected, and all the time cursing the jerks, while they were thrown to the earth with violence."[2]

The first form in which these spasmodic movements made their appearance was that of a simple jerking of the arms from the elbow downward. When they involved the entire body, they are described as something terrible to behold. The head was thrown backward and forward with a celerity that alarmed spectators, causing the hair, if it was long, "to crack and snap like the lash of a whip."[3]

[1] Davidson's 'History.' [2] McFerrin's 'Methodism in Tennessee.'

[3] Dr. Davidson, who relates this singular fact, felt it necessary to authenticate the statement by referring to eye and ear-witnesses of its reality. I remember to have heard my grandmother describe, when I was but a little boy, the same thing as occurring in a woman at a camp-meeting near her home in Tennessee, in 1810.

The most graphic description of the "jerking exercise" was written by the Rev. Richard McNemar, an eye-witness of the frenzy, as well as an apologist, believing it to be a display of Divine favour. In his 'History of the Kentucky Revival,' he says: "Nothing in nature could better represent this strange and unaccountable operation than for one to goad another, alternately on every side, with a piece of red-hot iron. The exercise commonly began in the head, which would fly backward and forward, and from side to side with a quick jolt, which the person would naturally labour to suppress, but in vain; and the more any one laboured to stay himself, and be sober, the more he staggered, and the more his twitches increased. He must necessarily go as he was stimulated, whether with a violent dash on the ground, and bounce from place to place like a football, or hop round with head, limbs, and trunk twitching and jolting in every direction, as if they must inevitably fly asunder. And how such could escape without injury was no small wonder to spectators. By this strange operation the human frame was commonly so transformed and disfigured, as to lose every trace of its natural appearance. Sometimes the head would be twitched right and left to a half-round with such velocity, that not a feature could be discovered, but the face appeared as much behind as before. Head-dresses were of little account among the female jerkers. Handkerchiefs, bound tight round the head, were flirted off with the first twitch, and the hair put into the utmost confusion; this was of very great inconvenience, to redress which, the generality were shorn, though directly contrary to their confession of faith. Such as were seized with the jerks were wrested at once, not only from under their own government, but from that of every one else, so that it was dangerous to attempt confining them or touching them in any manner, to whatever danger they were exposed. Yet few were hurt, except such as rebelled against the operations through wilful and deliberate enmity, and refused to comply with the injunctions which it came to enforce."

The same writer gives the history of a case of jerks as follows, and no case could illustrate more strikingly the nature of the affection.

A young man of a pious family, the son of a tanner, feigned sickness one Sunday morning to avoid going that day to camp-meeting. He kept his bed until he was assured that all the family, except a few negro children, had left the premises, and was much pleased at the success of his stratagem. As he lay quietly in his bed, his thoughts naturally turned to the camp-meeting in progress. The assembled multitude, excited, agitated, convulsed, rose up vividly before his mind. All at once, while occupied with the scene, he felt himself violently jerked out of bed, and dashed round the walls in a manner utterly beyond his control. Prayer, he remembered, was deemed efficacious in such circumstances, and he fell upon his knees in the hope that it would prove a sedative in his case. It turned out as he hoped, and he returned to bed, happy at finding the spirit exorcised. But the enemy soon returned; the jerks were as bad as ever, but were again allayed by prayer. Dressing himself, he now went to the tanyard, and set about currying a hide to occupy his mind. He rolled up his sleeves, and, grasping his knife, was about to commence the operation, when suddenly the knife was flirted out of his hand, and he was jerked violently backward, over logs and against fences, as before. Gaining relief by resorting once more to prayer, he ventured to resume his occupation, but was again seized with convulsions, and at last forsook the tanyard and betook himself to strong cries for mercy, at which he was found engaged by the family on their return from the meeting in the evening.[1]

Another characteristic example is related by a writer in the 'Gospel Herald':—[2]

A gentleman and lady of some note in the fashionable world were attracted by curiosity to the camp-meeting at Cane Ridge. They indulged in many contemptuous remarks on their way, about the poor infatuated creatures who rolled over screaming in the mud, and promised jestingly to stand by and assist each other in case that either should be seized with the convulsions. They had not been long on the ground looking upon the strange scene before them, when the young woman lost her consciousness, and fell to the ground. Her companion,

[1] Davidson, op. cit. [2] 'History of Methodism in U. States.'

forgetting his promise of protection, instantly forsook her, and ran off at the top of his speed. But flight afforded him no safety. Before he had gone two hundred yards, he too fell down in convulsions, "while a crowd flocked round him to witness his mortification, and offer prayers in his behalf."

These nervous disorders assumed many other grotesque forms besides those which have been described. The subjects often rolled over and over on the ground, or ran violently until worn-out with the exertion. Hysterical laughter was another modification. Instances of laughter were only occasional at first, but it grew, until in 1803 the "holy laugh" was introduced systematically as a part of religious worship. Sometimes half the congregation, apparently in the most devout spirit, were to be heard laughing aloud in the midst of a lively sermon. As the excitement grew, the infatuated subjects took to dancing, and at last to barking like dogs. McNemar says they actually assumed the posture of dogs, "moving about on all fours, growling, snapping the teeth, and barking with such an exactness of imitation as to deceive any one whose eyes were not directed to the spot."[1] Nor were the people who suffered so mortifying a transformation always of the vulgar classes: persons of the highest rank in society, on the contrary, men and women of cultivated minds and polite manners, found themselves, by sympathy, reduced to this degrading situation.

The "barks" were looked upon, at first, as a chastisement for remissness of duty, and the only way to escape them was to engage in the holy dance. But, from being regarded as marks of guilt, these wretched exercises came to be esteemed "tokens of Divine favour, and badges of special honour."[2] With these manifestations the insanity reached its height in about three years after it began to show itself.

It was one of the popular beliefs of the times that certain instincts or conditions of the system would avert these nervous attacks. Thus it was held, that a woman with a child in her arms, or conscious of approaching maternity, was in no danger. But there was no truth in the supposition. The maternal instinct, at least, had no protective efficacy. An instance is related where a woman mounted the stand, with an infant in

[1] Davidson. [2] Ibid.

her arms, for the sake of a better prospect, and that being suddenly seized, she fell backward, dropping her child. Some one fortunately saw the danger in time to seize and save the child before it fell to the ground.[1]

A large proportion of the members of every congregation had power to resist the convulsive tendency. In a great majority, no such tendency probably existed; but where there was a conscious impulse toward the convulsions it could be restrained by most persons before it had been yielded to too long. Dr. Blythe had but little of the disorder in his church. He discountenanced the wild enthusiasm from the beginning, and threatened to have any one who became convulsed turned out of doors. The religious frenzy soon began to abate when the clergy set their faces against the stormy exercises. Rev. Joseph Lyle, on the second sabbath in July 1803, preached in his church a significent sermon on "Order." The congregation had come together expecting the usual displays of feeling; but though some were angered by his doctrines, and some strove to promote the confusion of intermingled exercises, only a few "fell," and, altogether, moderation triumphed. This was the first sermon preached against the fanaticism.

It is a remarkable fact that, notwithstanding the intensity and duration of this nervous disorder, no instance is recorded in which permanent insanity resulted from it. Such results were to have been expected; insanity is mentioned by Edwards as having attended the excitement in New England, and it may be that reason was dethroned in some whose cases have not become matters of history. In a few years, after a sounder public opinion began to assert itself, instances of the disorder had become rare, but it was many years before the epidemic entirely ceased.

As to its nature, there was but one opinion among medical men from the beginning. All referred it to a derangement of the nervous system. Dr. Felix Robertson, of Nashville, described the affection in his thesis, published in Philadelphia, in 1805, as a form of Chorea. In some cases it took the form of that disease. In others it bore a stronger resemblance to epilepsy; while in a greater number it partook rather of the

[1] Davidson.

character of hysteria. It was eminently sympathetic in its nature, as has been so often remarked of these affections. The convulsions once started in a congregation spread quickly through it, until all the fit subjects were convulsed. Repetition greatly increased the proneness to the disorder, which was invited by the masses on the supposition that it was a true religious exercise.

These perverted muscular movements all come under the head of morbid reflex action. By the continued religious fervour, the central portions of the brain, the immediate seat of emotion and feeling, became inordinately excited. The impression, transmitted downward to the spinal cord, threw the muscles of voluntary motion into convulsions. Sensibility, which has its seat in the sensory ganglia, was generally annulled. When the hemispheres became involved, the subjects fell into a state of unconsciousness or coma. In this abnormal condition of the nervous centres, the bare recollection of the distressing scenes was sufficient in many cases to excite the convulsive movements. The former belong to sensori-motor actions; this last is an example of ideo-motor movement; instances of which are afforded by the act of vomiting, which may be caused by the recollection of disgusting sights or odours. The principle of imitation accounts for the rest. The great nervous centres, in multitudes of people, being in a state of polarity, any unusual exhibition of feeling would throw the more excitable into spasms; and the affection would then spread by sympathy, as hysterical convulsions and chorea are known to spread among girls at boarding-schools. And as fear has checked these, the epidemic convulsions were checked by reason and common-sense, and finally ceased under the law which limits all violent action.

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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(Continued from page 99.)

Gravimetric Methods.

Weight of Brain.—The amount of information upon this subject is already large, the number of enquirers in this field being legion. Before entering upon any such investigation, the student must make himself familiar with the various circumstances which modify brain weights. Some of these conditions have been summarised by Bastian.[1] They appear to be chiefly as follows:—

1. Length and nature of illness.
2. Mode of death (vascular engorgement favouring high weight).
3. Certain neuroses or epilepsy.
4. All conditions inducing sclerosis.
5. Atrophy.
6. Congenital micro- or megalo-cephaly.

The student must also take into consideration the relationship between weight of brain and the *age* and *sex* of the individual, as well as the *weight and height of body.*

Before weighing the brain, all fluid from the subarachnoidal space and ventricles must be drained off, and allowance be made for the membranes, which will scarcely exceed an ounce in weight. The plan adopted at the West Riding Asylum,

[1] 'The Brain as an Organ of Mind,' chapter xx.

however, is to first strip the brain of its membranes, to liberate the fluid in the ventricles, and weigh the whole encephalon. The membranes can afterwards be weighed if thought necessary. When the pia mater is firmly adherent to the cortex, it is as well to weigh the brain prior to stripping, as large shreds of the cortex are often removed in these cases. Precaution must be taken, however, by incisions, to release any accumulation of fluid in the subarachnoid spaces and meshes of the membrane, and, subsequent to weighing, allowance must be made for the pia mater and arachnoid. After the weight of the encephalon has been obtained, the cerebellum and pons Varolii must be removed by dividing the crura cerebri close to the pons Varolii.[1] A longitudinal incision is then carried through the median line of the corpus callosum from before, backwards, so as to separate the two hemispheres. The peduncular connections of the pons Varolii and medulla oblongata with the cerebellum are then divided, and the former separated from the medulla at its natural line of division. This is the method adopted at the West Riding Asylum, but for several years it has also been the custom to separate the frontal lobes from the remaining posterior part of the brain by an incision carried through the fissure of Rolando. The weights of these individual parts are then taken.

It will be found advisable to use the metric system in all our estimates of capacity, volume and weight, and all the standard brain-weights afforded by the elaborate tables of Tiedemann, Reid, Boyd, Wagner, and others should be expressed in grammes rather than ounces. Dr. Sharpey, after an elaborate analysis of brain-weights given by Glendinning, Sims, Tiedemann and Reid, supplies us with the following valuable results :—[2]

				oz.
Maximum	weight	of adult	Male Brain	65
Average	„	„	„	49½
Minimum	„	„	„	34
Maximum	„	„	Female Brain	56
Average	„	„	„	44
Minimum	„	„	„	31

[1] A modification of the plan is recommended in the section on the dissection of the brain as preferable when the question of weight is of secondary import (page 440).

[2] 'Elements of Anatomy,' 7th edit. vol. ii. p. 568.

The heaviest human brain on record has been described by Dr. Morris,[1] and was carefully examined at University College Hospital; the weight was 67 oz.

Compatible with ordinary intelligence, the lowest limit of the human brain as regards weight is, according to Gratiolet, 900 grammes, and, according to Broca, 907 for the female, and 1049 grammes for the male, brain.[2]

In connection with the weight of the brain in the insane, the student will find most valuable information in articles by Dr. Crochley Clapham, contained in the 3rd and 6th vols. of the West Riding Asylum Medical Reports. His observations embrace 1200 cases of insanity. Further information upon this subject may be obtained by reference to the following:—Sims, 'Medico-Chirurg. Trans.,' Vol. XIX.; Glendinning, 'Medico-Chirurg. Trans.,' Vol. XXI.; Tiedemann, 'Das Hirn des Negers,' Heidelberg, 1837; Reid, 'London and Edin. Mo. Jo. Med. Science,' April, 1843; Thurnam, 'Jo. Mental Science,' 1866; Wagner, 'Vorstudien,' 1862, 2^{e} Abh. pp. 93–95; Peacock, 'Mo. Jo. Med. Science,' 1847, and 'Jo. of Pathol. Soc.,' 1860; Boyd, 'Philos. Trans.,' 1860; Bastian, 'The Brain as an Organ of Mind,' 1880; Clapham, loc. cit. and 'Journ. of the Anthropolog. Inst.,' Vol. VII. p. 90.

Specific Gravity.—Researches upon the specific gravity of the encephalon are becoming more and more interesting with our knowledge of cerebral localisation and intimate brain structure. The late suggestive work of Danilewsky upon the relative amount of grey and white matter in the brain depends greatly upon the accuracy of the specific gravity for its correctness. The more important methods adopted are those of Drs. Bucknill, Sankey and Peacock.

Bucknill's Method.—Dr. Bucknill was the first who originated a satisfactory and ready method for estimating the specific weight of brain. The following is his detailed account of the process:—"The specific gravity of the cerebrum and cerebellum is ascertained by immersing a portion of each in a jar of water wherein a sufficient quantity of sulphate of magnesia

[1] 'Brit. Med. Journ.,' Oct. 26, 1872, p. 465.

[2] Quoted by Bastian, 'The Brain as an Organ of Mind,' 1880, p. 365.

has been dissolved to raise the density of the fluid to the point required, adding water or a strong solution of the salt, until the cerebral mass hangs suspended in the fluid without any tendency to float or sink, and then, by testing with the hydrometer, the specific gravity is thus found with great delicacy and facility, a difference of half a degree in the density of the fluid being indicated by the rise or fall of the substance immersed. The soluble salt is chosen, for its possessing no astringent or condensing action upon animal tissues."[1]

Specific Gravity Test.

Sankey's Method.—This process is the one which has been used most extensively by Dr. Crichton-Browne at the West Riding Asylum, and it is the method which I have myself invariably adopted. It appears to me in every respect highly satisfactory and simple. We require a series of cylindrical glass jars, such as the one figured, and a set of graduated hollow glass bulbs, which can now be readily obtained.[2]

These glass bulbs are accurately graduated, or rather marked with the specific gravity of the fluid in which they would hang suspended when immersed, neither tending to float nor sink. The glass jars are partly filled with water and then a concentrated solution of Epsom salts added to each, until its specific gravity is such that whilst one bulb floats, a bulb two degrees higher sinks. Thus a series of jars are filled as represented below.

Bulbs floating . . .	1030	1031	1032	1033	1034
Specific gravity of fluid =	1031	1032	1033	1034	1035
Bulbs sunk	1032	1033	1034	1035	1036

[1] 'Lancet,' 1852, vol. ii. p. 589.

[2] Such a set of graduated bulbs may be obtained of Mr. Stevenson, Philosophical Instrument Maker, No. 9, Forrest Road, Edinburgh.

The middle line represents the specific gravity of the fluid, and therefore of the portion of brain which tends neither to float nor sink, but to remain suspended wherever placed. It will occur to the student that the jars may be so graduated, that whilst one bulb floats, another, *one degree* higher, sinks, and hence that half a degree specific gravity may be indicated as follows:—

Bulb floats	1030	1031
Specific gravity of fluid and suspended body .	1030·5	1031·5
Bulb sinks	1031	1032

This is a degree of nicety, however, to which the use of the beads should not be pressed, and if required, should demand in preference the use of the 1000 gramme specific gravity bottle. A series of jars should be graduated from 1028 to 1050, so as to enable us to deal with white or grey matter of cerebrum, cerebellum and central ganglia. Occasionally still more dilute solutions will be requisite. A minute piece of white or grey matter is now raised by a scalpel and placed upon a perforated spoon or scoop, which is gently lowered into one of the jars of saline solutions, and the fragment of brain turned off the scoop and closely observed. If it sinks, it is of course of higher specific gravity than the fluid, and must be passed on to stronger solutions until, reaching one of its own density, the fragment remains stationary where placed. If, on the other hand, it floats, it must of course be moved to a solution of less specific weight, until the same conditions are obtained. Should the specific gravity beads be chosen by the student for this purpose, it would be well for him to attend to the following rules:—

1. After obtaining the specific gravity of any one portion of brain, repeat the trial with fresh portions at least two or three times, so as to ensure perfect accuracy.

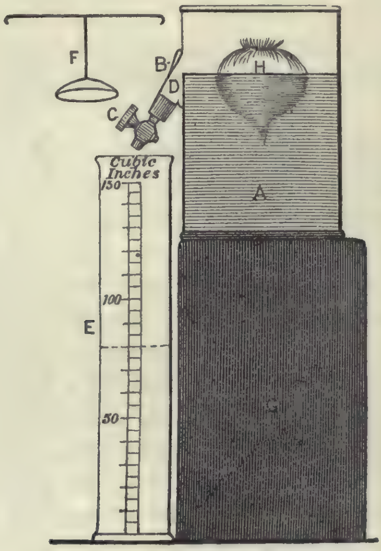
2. Examine the brain as soon as possible after death, wholly rejecting such as show the least evidence of commencing decomposition.

3. Keep the jars covered with glass squares in a cool room, so as to avoid dust and evaporation as far as possible.

4. Before commencing any series of observations, note the

position of the bulbs, any tendency of the lower bulbs to float from evaporation of the solution being rectified by the addition of water.

Stevenson's Displacement Apparatus. — A simple and most reliable apparatus has been devised by Mr. Stevenson for estimating at the same time both the bulk and specific gravity



Stevenson's Displacement Apparatus.

of large irregular bodies by the principle of displacement. The apparatus consists of a jar, A, fitted with a large drooping tubulature, B, with stopcock attached. A metal gauge, D, having a thin horizontal straight-edge inside the jar, determines the exact water-level, and over it the surplus water flows. The other jar, E, is graduated on the sides into cubic inches, avoirdupois lbs., oz., and thousandth parts of a gallon. The packing case, G, serves as a stand for the jar. In taking the specific gravity of the brain, or any body a little heavier than water, the jar must be filled with a solution of salt or sulphate of magnesia up to the level of the gauge, the stopcock being closed. The brain is then immersed in the fluid, in which it ought to float, and the fluid immediately rises in the jar; let it come to rest; open the stopcock, and

the quantity displaced will flow into the graduated jar and represent the weight-indication of the brain (since any floating body displaces exactly its own weight of the fluid). The stopcock is now shut, and the brain depressed by the brass piece, F; and when the water has again come to rest, open the stopcock and receive the further displacement in the jar E. The whole bulk of displaced fluid read off the graduated jar indicates of course the volume of the brain. To obtain the specific gravity, multiply the weight-indication by the specific gravity of the fluid and divide by the volume-indication. I need scarcely indicate to the student the simplicity and value of this method, whereby the *weight*, the *volume* and the *specific gravity* of the brain may be simultaneously obtained.[1] The apparatus, as figured, is somewhat smaller than what would be required for brain.

In estimating the specific gravity of the encephalon, the student will soon appreciate the fact that the specific weight varies not only in the cerebrum, central ganglia and cerebellum, but that variations occur over different regions of the cerebrum, suggesting the importance of a comparative examination by this method of the various cerebral convolutions. By cautious manipulation he will also be able to show that the specific gravity of the cortex varies with its depth. Dr. Sankey observes that the specific gravity diminishes in the ratio of ·001 for every twenty-four hours after death. This must, however, be greatly modified by the temperature of the surrounding atmosphere, and other conditions favouring putrefactive changes. The following embrace the more important results obtained by different observers, and they will serve as a useful guide to the student in his prosecution of similar observations:—[2]

Average specific gravity of whole	Encephalon	1036	(Bucknill.)
,, ,, ,, ,,	Cerebrum	1030–'48	(Aitkin.)
,, ,, ,, ,,	Cerebellum	1038–'49	(Aitkin.)
,, ,, ,, ,,	Grey matter	1034	(Sankey.)
,, ,, ,, ,,	White matter	1041	(Sankey.)
,, ,, ,, ,,	Central ganglia	1040–'47	(Aitkin.)

[1] This apparatus is sold by Mr. Stevenson, 9, Forrest Road, Edinburgh.

[2] References to work done in the Specific Gravity of the Brain:—Bucknill, 'Lancet,' vol. ii. 1852, and the 'Medico-Chirurg. Rev.' 1855; also 'Psychological Medicine,' Bucknill and Tuke, 3rd edit. pp. 520 and 587; Sankey, 'Brit. and

Proportion of White and Grey Matter in the Brain.—The specific gravities of the grey and white matter, together with that of the whole brain, have been applied to solve the very important problem of the relative percentage of grey and white matter in the human brain. Danilewsky has lately published his results and method of procedure, an account of which may be seen in the 'Centralblatt f. d. Med. Wissenschaften,' No. 14, 1880.[1] The formula given by him is as follows:—

$x = \frac{Pb\,(p - a)}{p\,(b - a)}$ where x is the *quantity* of grey or of white matter, p the *specific gravity* of the whole brain, a of the grey and b of the white substance, and P is the *weight* of the whole brain. He gives as the results of one series of experiments a percentage for the grey matter of 37·7 to 39· against a percentage of 60·3 up to 61· for the white substance. By taking the average depth of grey matter it is of course an easy matter to obtain the superficial area of the brain. Wagner's researches upon the superficial area of the brain are of interest here (quoted in Quain's 'Anatomy,' vol. ii.). The student, however, must be warned against regarding results so obtained as other than merely *approximate*, as it is next to impossible to exclude the numerous fallacies to which the estimation of average specific gravity is liable. Thus the specific gravity of the grey matter is known to vary with its *depth*, with its *local distribution or area*, with the amount of *vascularity* of the tissue and *time after death*. Beyond this, the very cases in which the pathologist would be interested in estimating the relative proportions of grey and white matter are those which are subject to such diffuse and local change in consistence that an average specific gravity for either white or grey could not be attained with any degree of exactness. For comparative investigation of healthy brain, the process adopted by Danilewsky promises to afford valuable results as long as the greatest care is observed to exclude fallacies.

Removal and Dissection of the Brain.—It may be useful here

For. Med.-Chir. Rev.' 1853; Peacock, 'Trans. Pathol. Soc. of London,' 1861–2; Aitkin, 'Science and Practice of Medicine,' 1865, vol. i. p. 265.

[1] Vide Abstract by Dr. Geoghegan, 'Journ. Mental Science,' p. 437, Oct. 1880.

to review the course to be pursued in removing and preparing the various parts of the brain for coarse examination, and the following method is recommended as in every way the more reliable and satisfactory one.

In the first place, the head should be so supported as to command a good light and a full and satisfactory view of the brain *in situ.*

In the next place, never neglect the rule of opening the skull and examining the brain *prior to opening the thorax,* and dividing the great vessels.

Cranial Membranes and External Aspect of Brain.—The skull-cap having been removed *sec. art.* we proceed as follows:—

1. Examine upper surface of dura mater in its relationships to the skull.

2. With a forceps raise a fold of dura mater anteriorly, run the scalpel through it, and carry the blade along either side on a level with the sawn edge of the cranium far back towards the occiput, stopping short of the middle line.

3. Reflect these lateral halves of the membrane towards the middle line, observing the condition of the large superficial veins running into the longitudinal sinus.

4. Replace the dura mater, and with a curved bistoury open up the longitudinal sinus in its whole length back towards the torcular herophili, noting the condition of the parts.

5. Divide the connection between dura mater and crista galli, and seizing the anterior end of the falx cerebri forcibly, draw the membrane backwards, dividing the junction of the superficial veins in this course, and thus expose the surface of the hemispheres.

6. With a good light thrown on the subject, and, if requisite, the aid of a hand lens, observe the appearances presented at the vertex, noting the condition of the vessels, membranes and general conformation of the brain. It is absolutely necessary to pay attention to this point, as the appearances presented *in situ* are often greatly modified or wholly lost upon removing the brain at a later stage.

7. Gently raise the tips of the frontal lobes from the orbital plates, carefully removing the olfactory bulbs with the brain, and then using a little gentle traction, the optic nerves are exposed

and divided close to their foramina. With the same blunt-pointed curved bistoury divide successively the infundibulum, carotid arteries and third nerve. A good view is thus afforded of the tentorium, which should be divided along its attachment to the ridge of the temporal bone, dividing at the same time the fourth nerve. The base of the brain is now exposed, and the fifth, sixth, and seventh nerves readily divided, after which the blade is passed down into the vertebral canal on either side the medulla, and cutting forwards, it severs the vertebral arteries, the eighth and ninth pair and spinal accessory nerves. A sweep of the bistoury across the front of the cord liberates the brain, which can be now raised out of the cranial cavity with ease.

8. The next procedure is to place the brain base upwards in the skull-cap, the latter being conveniently steadied by any simple contrivance. This enables us with less sacrifice to the appearances at the vertex to study the important region of the base. In doing so, follow out the instructions already given, taking the parts in the following order :—

a. Arrangement and appearance of the great vessels at the base.

b. Condition of the membranes and subjacent gyri.

c. Condition of the various cranial nerves.

d. Open up the fissure of Sylvius on either side, tracing the large arterial branches upwards; also examine nutrient supply to the basal ganglia at anterior and posterior perforated spots.

e. Strip the base of its membranes, noting adhesions, &c. &c.

9. The cerebrum may now be turned out upon the dissecting tray, with vertex uppermost, and the membranes carefully examined and stripped. Take note of the arrangement of the gyri, the presence of superficial lesions, general consistence, and colour.

10. The dura mater and its sinuses at the base of the skull may be now examined, completing thus the examination of the cranial membranes and the external aspect of the brain.

(*To be continued.*)

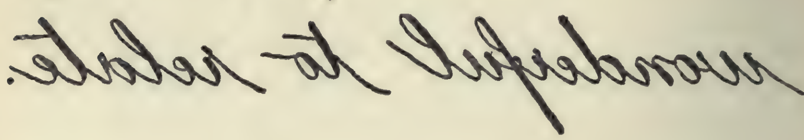
ON MIRROR-WRITING AND ITS RELATION TO LEFT-HANDEDNESS AND CEREBRAL DISEASE.

BY WILLIAM W. IRELAND, STIRLING.

BUCHWALD and Erlenmeyer have directed attention to what they call Spiegelschrift or mirror-writing, because, like the impression of a letter taken upon blotting-paper, it can be most easily read by those not used to it in a mirror, where the reflected image takes the appearance of ordinary writing. This inversion of our written characters is sometimes done as a species of puzzle for amusement or curiosity; but I have lately met with several instances where it was seriously produced, apparently as an imitation of ordinary writing.

E. M. was a paralytic imbecile girl. She was believed to have been hemiplegic on the right side from birth, though the weakness was not decidedly noticed till she was six months old. When admitted into the Larbert Institution in 1875, aged 7 years, she was able to say a few words rather indistinctly. She was found to be subject to occasional visits of epileptic vertigo, and more rarely epileptic fits. She was helpless with the right arm, but able to halt along with the right leg. The outline of the head was rounded, or slightly bulging on the left side above the ear. The girl was active in disposition, mirthful, and somewhat mischievous. About eighteen months ago on the governess commencing to teach her to write, which was done by getting her to copy a lithographed line at the top of the page, the girl formed the

letters with the left hand from right to left in mirror-writing thus—



L. N. (aged 14), genetous imbecile girl, of considerably greater intelligence than the first case, is scoliotic in spine, and deformed in the lower limbs through rickets. The head is of good size and regular shape, save that it is somewhat flattened at the occipital region. There is a bulging behind and above the right ear. The palate is rather unsymmetrical in shape, and some of the teeth are irregularly placed $\frac{1\,5}{1\,5}$. There are eight on the right side of the upper jaw, and eight on the left side of the lower one. She used to be left-handed. This girl also began to write in mirror-writing with her left hand, but was interdicted, and has now entirely given it up. She has been gradually broken from using the left hand, and sews pretty fairly with the right. When I asked her to give me a specimen of the mirror-writing, I found that she could only do it with her left hand. Apparently she can write from right to left with about as much ease as from left to right, but cannot now read it so well; she reads from her lesson-book with some effort words of two syllables, and sews pretty fairly with the right hand. Though she speaks freely on simple subjects, she cannot make any explanation as to the directions which she gives to her writing with either hand; but one cannot expect any analysis of a mental process or complex action from an imbecile girl.

There are two idiot boys in the school who are forming pothooks from right to left, being left-handed, so that in time they would teach themselves mirror-writing.

I have consulted several experienced superintendents of Training Schools for Idiots who have seen no instances of mirror-writing with imbeciles; but Mr. Millard, the Superintendent of the Eastern Counties Asylum, Colchester, has

kindly communicated to me the description of one case. A. B. W., imbecility believed to be congenital, admitted when 12 years old. Teeth pretty good, "spine and limbs partially paralysed," choreic, can walk and run, dress and feed himself. He can do simple sums in subtraction, multiplication, and division; can read, and possesses moral and religious sense; his judgment is defective; knows his right hand from his left. "He writes backward with his left hand, so that it is only legible by turning the paper round or by a mirror."

A friend of mine who had seen the mirror-writing of the imbecile girl was struck at finding the same inversion in one of his own pupils. He was left-handed, and as teachers think it their duty to compel left-handed children to use their right, the boy, finding this difficult, when the teacher was not looking secretly wrote with his left hand, and the result was a page of mirror-writing, which the boy apparently thought was a copy of the lithograph.

The boy is 13 years of age, pale, thin and flabby. He is infirm in the legs to such a degree that it might be described as a minor degree of paraplegia. I thought the right leg was weaker than the left, but the boy did not seem to be sure of this himself. The head is somewhat small, but quite as symmetrical as is usually met with; the contour slightly fuller on the left side above the ear. Out of school he still uses the left hand. The teacher describes the boy as rather intelligent, and getting on well at his lessons. On being requested to copy a passage out of a book in mirror-writing, he very soon returned with it fairly copied. I asked him, "Did you write this with your right or your left hand?" at which he said, with some hesitation, that he did it with his right. I told him nobody would be angry with him; when he confessed that he had written it with his left hand, as we had asked him for a specimen of the writing and he could only do it with the left hand. He could read the mirror-writing fluently. It is perplexing that any one should in copying a line lithographed at the top of the page imagine he was correctly reproducing it when he was writing it in an inverse direction.

For example, if any one were told that he must write the

word "wonderful" from right to left, he would commence with the *l*, and trace the letters backwards; while these two pupils not only wrote from right to left, but they inverted the image of the word, so that while the *w* of the copy was on the left, in their imitation it appeared on the right, as if they had scratched on a pane of glass, and turned it and read it on the opposite side. This, of course, is different from ordinary handwriting from left to right, such as was practised by the Hebrews and Etruscans, and in the modern Arabic letters throughout the Mahommedan world. In their manuscripts or lithographs the lines begin at the free side of the page and run to the left, but then the Arabic letters are naturally adapted to be traced in this way, and indeed it would be difficult to form them in any other. Familiar with this writing by my residence in India, I am of the opinion that if it is more difficult to read than the English characters, this is not because it runs from right to left, but, owing to the suppression or uncertain quantity of vowels, the writing is so little phonetic that it needs a knowledge of the language ere one can read a Hindustani or Arabic book. A clerk cannot copy Arabic writing so quickly as English, but this is owing to the nature of the characters, which are more numerous, and most of them having an initial, medial and final form.

I have been told by one who practised mirror-writing for amusement, that it is easier to trace with the left hand, and the following experiment, made by my friend the teacher, will show that there is a physiological tendency with left-handed children to fall into mirror-writing. He took a class of sixty boys and girls, and told them all to write their names with their left hands. All copied as well as they could, writing from left to right. Some two girls and three boys wrote in mirror-writing. These were found to be all left-handed, and the only left-handed in the sixty. It did not appear that these children were conscious that they were writing in an inverse direction different from the rest. The left-handed children went to work instantly without any perplexity, traced their letters better than the other children.

Miss C., the teacher in a public school, took 134 children of the junior division, and, getting the assistance of a

colleague, separated them into small divisions, gave them pencil and paper, and told them to write with their left hands, and not to look on one another's papers. Apparently there were six children known to be left-handed or to have a tendency to use the left hand, and three of these wrote in mirror-writing, and none else. In one experiment it was found that a man who tried to write a few Hindustani words in Arabic characters with his left hand, unconsciously traced the letters from the left in mirror-writing.

Dr. Erlenmeyer, in his interesting pamphlet on the Physiology and Pathology of Writing,[1] observes that it seems to be easier to use the arms in a centrifugal direction, the left from the right and the right from the left, the motions not being hindered by the trunk of the body, and that where ease, elegance and security are needed the movements of abduction are always performed. He gives turning a hand-mill, striking a lucifer match, and executing the most brilliant passages on a piano as examples, and assures us that he could easily give more of the kind. In that case his instances do not seem to be well chosen. I have been assured that many of the most striking passages on the piano are performed both to and from the centre, and some exercises requiring skilful execution are certainly done in a centripetal manner; using the sling, bowling and batting in cricket are examples; and in fact, whether in fencing, swimming, sewing, or other actions, movements must be made both from and towards the centre of the body. Nevertheless, taking everything into consideration, it appears true that most actions requiring skill in their performance are done easiest by the arms in a centrifugal direction.

Dr. Wilbur of Syracuse (N. Y.) has kindly sent me specimens of the performance of a man who could write the same words with both hands at once, the right hand in the usual way, the left in the mirror-writing; but as he could also do the same feat with both hands moving from left to right in ordinary text, it seems to be more a piece of sleight-of-hand than any obedience to a physiological tendency. Dr. Wilbur

[1] 'Die Schrift, Grundzüge ihrer Physiologie und Pathologie von Dr. Albrecht Erlenmeyer;' Stuttgart, 1879.

mentions the case of a left-handed child who, when beginning to read, asked his father what "efiw" was. On being told that there was no such word, the child brought his book and pointed out the word "wife." The boy for some time after made similar mistakes. Such inversions not unfrequently occur in teaching imbecile children to read; they will call "no," "on," or "was," "saw." At Larbert we generally taught them small words before teaching them the letters.

Buchwald, in a contribution to the Berlin 'Klinische Wochenschrift,' three years ago, gave the case of a man of 45, who presented the ordinary symptoms of apoplexy with hemiplegia of the right side. After the somnolence had disappeared, which for some days followed the attack, it was found that he was aphasic. He was induced to try writing with the left hand, as he could not do it with the right. He wrote in a very skilful manner his name in mirror-writing from right to left, as well as the numerals from 1 to 10, except the figure 8, which he had forgotten. The inverse direction of his writing was pointed out to him, but he could not be induced to try writing from left to right. His name and some figures being written out and held before him, he copied them awkwardly, but again fell into the mirror-writing. After a time he traced the numbers 1, 2, 4, 6, 8, and 9 correctly, but gave 3, 5, and 7 in mirror-writing. He was asked to multiply a few figures, and the ciphers were correctly put down for him; he wrote the sum from right to left. In this case he must have multiplied the numbers in his mind and then recorded the result in mirror-writing. The patient remained about six months in the hospital at Berlin, during which time, though the aphasia, agraphia, and alexia disappeared, the tendency to mirror-writing still persisted. He gave himself great trouble in trying to copy writing from left to right; he said that he could not perform it in this direction with the left hand; when he again had the use of the right hand he would do it correctly. In trying to trace the letters from left to right he was obliged to use the half-paralysed right hand to help the left, otherwise the operation miscarried. The 5 was the most difficult to form. Even with the right hand he traced the cipher in mirror-writing, at least he could not manage the hook of the 5 otherwise.

The best known example of a change from right-handed to left-handed writing, Dr. Erlenmeyer tells us, is that of the MS. of the 'Codex Atlanticus' of Leonardo da Vinci, in the Ambrose Library at Milan. It was generally said that in adopting this singular style of writing Leonardo wished to preserve his work from the eyes of superficial readers; but we can now give another explanation. There is a diary in the National Library at Naples, of the priest Antonio de Beatis, who in 1517 travelled in the train of the Cardinal of Arragon, through Germany, the Netherlands, and France. The Cardinal visited Leonardo da Vinci, who passed the last years of his life in the neighbourhood of Amboise, in a villa given to him by Francis I. De Beatis remarks of the famous artist, in his journal, "that nothing more of value in painting could be expected of him, as he had paralysis of the right hand." It would appear from this that Leonardo da Vinci, being unable to use his right hand, wrote with his left, and fell into the practice of writing from right to left, in obedience to a tendency which we have sought to illustrate.

It may be asked, is the image or impression, or change in the brain-tissue from which the image is formed in the mind of the mirror-writer, reversed like the negative of a photograph; or if a double image be formed in the visual centre, one in the right hemisphere of the brain and the other in the left, do the images lie to each other in opposite directions, e.g. C on the right side and Ɔ on the left side? We can thus conceive that the image on the left side of the brain being effaced through disease, the inverse image would remain in the right hemisphere, which would render the patient apt to trace the letters from right to left, the execution of which would be rendered all the more natural from the greater facility of the left hand to work in a centrifugal direction. Moreover, when one used the left hand to write, there would probably be a tendency to copy the inverse impression or image on the right side of the brain. Probably it would be well to collect more examples of mirror-writing, and to study them carefully before indulging in speculations; but it seems to me that there are grounds for suspecting that in some of the cases the mental image was really reversed, as well as the copy reproduced on the paper.

PERVERTED SEXUAL INSTINCTS.

BY DR. JULIUS KRUEG.

Physician to the Private Lunatic Asylum, Ober Döbling, Vienna.

During the last decade but one there appeared in Germany, at first under the pseudonym "Numa Numantius," but afterwards under the full name of an old Hanoverian official, Karl Heinrich Ulrichs, a series of writings[1] which attracted considerable attention. The writer, who was well known in political and legal circles as the author of various learned works, made a very remarkable assertion. He asserted that a great many men, "as a result of their inborn nature, felt themselves drawn by sexual desire to male individuals exclusively." Towards women their feelings were either indifferent, or actually repellent. Ulrichs named such men "Urnings." "The Urning," he says, "is a sport of nature; his bodily structure is that of a man, but his sexual instincts are those of a woman." Ulrichs sought to explain this anomaly by the supposition that a woman's soul dwells in a man's body ("anima muliebris virili corpore inclusa"), a possession which dates from the earliest embryonal period, before the differentiation of the sexual organs.

Westphal, influenced, it is fair to presume, by these writings, agreeing as they did with some facts that were even at that time known to the profession, in 1869 published the history of two cases[2] which were characterised by an inborn, morbid, unconquerable sexual desire towards individuals of the same sex. The first case is a very typical example of the affection, the second does not absolutely exclude another interpretation. Westphal defined the affection as "a congenital perversion of

[1] 'Inclusa' and 'Vindex,' 1864; 'Formatrix,' 'Vindicta,' and 'Ara spei,' 1865; 'Gladiusfurens,' and 'Memnon,' 1868; 'Incubus,' 1869.

[2] 'Arch. f. Psych.' Bd. ii. pp. 73–108, 1869.

the sexual instinct, with retained consciousness of the morbid nature of the condition," and he named it "contrary sexual instinct" (*conträre Sexualempfindung*), a symptom of a neuropathic (psychopathic) constitution.

This paper of Westphal's was followed in the course of the next few years by a number of others. Schminke[1] and Scholz[2] each published a case; and Gock[3] and Servaes[4] two cases each, but of Gock's cases only one is quite satisfactory. Westphal[5] reported a third case; Legrand de Saulle[6] a case; and Stark[7] four cases, to one of which, however, some uncertainty attaches. Krafft-Ebing[8] also published a case, and gave a *résumé* of the cases previously recorded, along with a systematic account of the various abnormal developments of the sexual instinct. At a later period there followed a case by Tomassia Ariggio,[9] and another somewhat doubtful case by Kelp.[10]

Without counting the cases published before Westphal's first paper, although they might very properly be included, and omitting those among recent cases that are not altogether satisfactory, there are now on record thirteen well-established cases of this affection; and to these the case that I am to relate will add a fourteenth. It is true I have seen the patient three times only, but from the information he has given me about himself I have little doubt as to the case. Moreover, and to this I attach particular importance, his narrative gave me the impression of being a truthful story—a

[1] Ein Fall von conträrer Sexualempfindung, 'Arch. f. Psych.' Bd. iii. pp. 225–226, 1872.

[2] Bekentnisse eines an perverser Geschlechtsrichtung leidenden, 'Vierteljsch. f. ger. Med.' Bd. xix. pp. 321–328, 1873.

[3] Beitrag zur Kenntniss der conträren Sexualempfindung, 'Arch. f. Psych.' Bd. v. pp. 564–574, 1875.

[4] Zur Kenntniss von der conträren Sexualempfindung, 'Arch. f. Psych.' Bd. vi. pp. 484–495, 1876.

[5] Zur conträren Sexualempfindung, 'Arch. f. Psych.' Bd. vi. pp. 620–621, 1876.

[6] Quoted by Krafft-Ebing.

[7] Ueber conträre Sexualempfindung, 'Allg. Ztsch. f. Psych.' Bd. xxxiii. pp. 209–216, 1877.

[8] Ueber gewisse Anomalien des Geschlechtstriebes, &c., 'Arch. f. Psych.' Bd. vii. pp. 291–312, 1877.

[9] Sull' inversione dell' istinto sessuale, 'Riv. Sperm.' 1878, pp. 97–117.

[10] Ueber den Geisteszustand der Ehefrau C. M., 'Allg. Zeitsch. f. Psych.' Bd. xxxvi. pp. 716–724, 1880.

candid confession. His experiences were not disclosed to me under the pressure of a formal inquiry, and with an endeavour to palliate an offence against morality by making it out to be the result of a disease. They were not the disclosures of an inmate of a prison or a lunatic asylum, who associated only with persons of his own sex, and whose sexual onslaughts, committed *faute de mieux* on his own sex, might be regarded by himself and others as the result of a morbid impulse. They were the confessions of a man, labouring, certainly, under great mental excitement, but totally uninfluenced by any external pressure, and they were called forth solely by his desire for support in the conflict he was waging against himself.

On one of the last days of April, a young man burst into my consulting-room and begged me to give him some medicine quickly, for he was afraid he was about to have a 'stroke.' After a few inquiries, I gave him tincture of digitalis, which so far allayed his excitement that he was able, after a while, to relate to me the history of his illness; only, however, in broken fragments, which were in part supplemented and corroborated by his brother, who accompanied him. The substance of his statements to me on this and the two other occasions on which I saw him is contained in the following narrative:—

Herr N. belongs to a neuropathic family. His mother, who is dead, was, to say the least, hysterical (she was very "nervous," and at times could not bear to look at "anything pointed"); a sister is similarly affected; and a brother shot himself three days before Herr N.'s first visit to me. The patient himself, apart from his perverted sexual feelings, suffers from nervous symptoms; but it will be more convenient to return to these at a later period.

Herr N. remembers that when only six years old the sight of naked men in a bath gave him a peculiar pleasure, which, at that time, he knew not how to interpret, but which he now recognises to have been a sexual sensation. At the age of nine, he got a fright from a snappish dog, as a result of which he suffered during the next five years from nervous disorder, which manifested itself principally in his frequently

starting up and speaking during his sleep. Convulsions he never had. He was sent to the country on account of the delicacy of his health, and so came to spend his school-days away from Vienna in a monastical institution for convict boys. There he became acquainted with the practice of onanism, which was usually performed reciprocally. At this time he conceived an extravagant fondness for one of his "friends," in which at last sexual desire and jealousy came to play the same part that they ordinarily do in love affairs. He found no pleasure in the occupations and sports of his comrades, and, as a consequence, took little part in them.

On his return to Vienna he devoted himself to a mercantile life, and he now, in company with his brother, conducts one of the best establishments in the city for ladies' dresses and millinery. Ladies' bonnets are his particular speciality, and as he possesses singular taste and skill in designing new shapes and arrangements, his services are of the utmost value. He makes several journeys to Paris in the course of the year in connection with his business, and he works at home, assisted by twenty to thirty women, with great enthusiasm. His brother has the management of the ladies' *toilette* department; but in this, too, Herr N. interests himself as an "amateur."

Herr N. lives under very favourable pecuniary circumstances, and he is now thirty-three years old; it is natural, therefore, to suppose that he must not unfrequently have been reminded that he ought to marry. And yet he feels no longing desire to have children and to found a family. On the contrary, he has an insuperable abhorrence of sexual connection with women. His brother, years ago, took him on one occasion to a brothel, but the visit was in vain; he ran from the place in disgust. He spends his days among women, and he has often invoked the aid of his imagination to induce a normal sexual desire; many a time, indeed, he has thought he has succeeded, but the next moment all his old horror has returned. Once he was almost engaged to a lady, but his insurmountable repugnance was not to be overcome, and the match was broken off before it was very seriously entered upon.

Whilst he has felt this dislike to sexual intercourse with the

opposite sex, he has continued to practise onanism until quite recently, both alone and with other men. One of his old schoolfellows came to his house, and he also associated with two new acquaintances. He confirms the statement, repeatedly made by others, that individuals affected with this abnormity are able to recognise one another. Even his imagination tends to dwell on the male sex only, and that, too, though he does all that he can to direct it towards the opposite sex. In his dreams, it is men who appear, or women who, at the last moment, turn into men.

Herr N. has again and again intended to conform to the natural use, and lately has so far carried out his resolve as to leave off all intercourse with men. But he has since experienced a constantly increasing mental irritation, for he has a very strong sexual appetite, and he has now no means of gratifying it. Moreover, he has lately undergone mental irritation of another sort. A brother, a pensioned officer, living with the rest of the family, was the subject of disapprobation in more ways than one. He was poor and needed money, and he was ill, and, in consequence, peevish and irritable. Three days before, after a scene with the before-mentioned hysterical and, we may add, quarrelsome and niggardly sister, he had shot himself. Herr N., in fact, had just come from the mass that had been said for him.

Herr N. complains also of various nervous sensations, which the reading of a popular medical work has made him believe are the premonitory symptoms of a grave nervous affection. Thus he has a peculiar twitching in the nape of the neck, which he is convinced is the commencement of tetanus. He has inherited the fear which his mother had of everything pointed; for instance, he dare not put his hand into a dish containing pins, frequent though the occasions for doing so are in his business. He also complains of losing sometimes the power of controlling his thoughts. Ideas present themselves which he is unable to banish (*Zwangsvorstellungen*). Thus there are times when, notwithstanding his inward strivings against it, his highest and most noble thoughts are burst in upon and jostled by the very lewdest and basest of ideas. When asked for a more specific example, after a long

delay he whispered into my ear that even during the mass for his brother he had felt compelled to think of a combination of the Host and the anus of a dog; and, as he is a believing Catholic, it may be conceived how horrified he is at such thoughts.

Herr N. is a young, rather small-made man, of middle size, and with normal genital organs. Although thirty-three years of age, he has a very sparing growth of beard, which he carefully removes. In dress and demeanour he is rather affected. During the narration of his case he walked up and down the room in an excited manner, not forgetting, however, to survey himself in the mirror as often as he passed it. His speech and gesture are strikingly theatrical.

A month after his first visit, Herr N. returned, and surprised me by stating that he had been in love for some time. It was soon apparent that the story was merely a creation of his imagination. The lady to whom he referred lived in Paris, and he had not seen her for more than six months; indeed he had only seen her a few times in all, with long intervals between. The excitement which he exhibited at his first visit had disappeared in a few days under the use of infusion of digitalis, but the sexual passion had again become strong, and he besought a remedy for it. He complained, too, of a kind of vertiginous attack; he had had some difficulty in getting from his house to the nearest cab-stand, and could now scarcely trust himself to walk. I assured him that he need have no fear on this score, and I prescribed for him bromide of potassium.

Fourteen days later he paid me a third visit. He had had no return of the vertigo, but he again complained of the twitching in his neck. The sexual feelings had moderated somewhat; but he expressed a doubt whether, after all, it was possible for him to find the natural way, and whether it would not be much more practical for him simply to revert to his old way of living, since the struggle otherwise seemed endless.

The object of my treatment has been, on the one hand, to strengthen his nervous system, and, on the other, to tone down his sexual desire so as to bring about a longer interval between the acts of sexual gratification. Only after

succeeding in this shall I attempt to direct his desire into the natural channel. Shall I succeed? I confess I feel less hopeful even than my patient.

In the case I have just reported, the fulness of the patient's confession, which bore, at all events in its main parts, the impress of truth, leaves little to be desired. It is different with the case I am now to relate. Here I can only conjecture that we have the same perversity, which has never got beyond a platonic stage. For this reason I cannot claim for it a place among the well-established cases, nevertheless I shall put on record what little I have been able to ascertain in regard to it.

The patient was a maidservant, twenty-five years old. Little is known of her previous history, except that she changed her place with extraordinary frequency, until she obtained her present situation with four young ladies (orphan sisters), with whom she has been for three years. And now she vows she will throw herself into the Danube if she receives notice to leave. She says she cares nothing for men, and other circumstances confirm this, for she is such a well-favoured woman she would certainly have long since found a lover had she cared for one. She is, however, passionately devoted to her ladies, and will cry all day if she thinks one or other of them has looked black at her, going about moaning that Miss does not want her any more, &c. She is a tolerably big woman, with strong but pretty features, not at all unwomanly in appearance, and, as far as can be judged, normally developed.

If we are to form an opinion merely from the number of published cases—fourteen in twelve years—we shall be obliged to conclude that a perverted sexual appetite is a very rare phenomenon. And yet the cases of pæderastia that again and again rise into public notice seem to indicate that it is by no means unfrequent; and Ulrichs, who has gone into the matter statistically, computes that one man in every 500 is an "Urning." My authority, too, whom I directly questioned on the subject, when he told me that "Urnings" could recognise one another, regards these figures as substantially correct. But, granted that they are excessive, and that the majority of the cases are simply cases of pæderastia, there still remain a

considerable number which are, in all probability, instances of perverted sexual appetite. It is difficult to obtain any definite information as to the frequency of this condition in women. Of the fourteen cases hitherto published, twelve occurred in men, and two in women. It must not be forgotten, however, that it is even easier for women to escape detection than men. If we group together all the cases published since Westphal's first paper, including the doubtful cases and those in which the perverted sexual instinct was only transient, we shall find that men were the subjects of the affection fourteen times, women eight times. Westphal has already drawn attention to the various practices that obtain among different peoples in this matter, and shown that light is thrown on them by the facts of perverted sexual instincts. It is clear that the perversion occurs both in civilised and uncivilised nations. I have no intention to enter at any length into historical or ethnographical considerations, but I may just mention the boy-loves of the Greeks and their Lesbian-loves, a parallel to which exists in the history of other peoples. It is, then, at least probable that these practices are, in part, attributable to the anomalous development of the sexual instinct.

In the well-defined cases, there has almost always been evidence of an inherited psychopathic, or, at all events, neuropathic constitution. This taint has manifested itself in all degrees, from slight neuropathic affections on the one hand to the graver forms of mental disease on the other.

But since, as yet, a comparatively small number of cases have been described, and these purely from a psychopathic aspect, it is not at all impossible that we shall find it difficult in some cases to establish the existence of a neuropathic diathesis. This, from a forensic point of view, is a matter of great importance.

Signs of degeneration (*Degenerationszeichen*) were frequently met with in these cases, and the question arises whether the perverted sexual instinct is not in itself, isolated though it be, a sufficient indication of degeneration. Biologically considered, there cannot be a doubt that an impulse so adverse to propagation must be a mark of degeneration.

The sexual organs, and the so-called secondary sexual

characteristics, were normally developed in all the cases. I make no exception of a case where there was phymosis. Herr N. tells me that his friend had a phymosis; indeed, the condition seems a common one in cases of this kind. Neither do I attribute much importance to the scanty development of beard in Herr N.'s case. A striking approach to the customs and habits of the opposite sex was frequently noticed, and several times the sexual instinct became active at a remarkably early age, or remained unusually strong at a late age. We need scarcely remark that persons with perverted sexual instincts, but very weak sexual desire, will not come so readily into conflict with the moral sense of themselves or others as those with heightened sexual desire, and will therefore the more readily escape observation.

The feeling of aversion to the other sex was general, but it did not in every case prevent occasional intercourse. Exceptions, in a measure, prove the rule, and in this respect the case reported by Scholz is very instructive. A man married in the hope of getting rid of his perverted sexual desire, but he could only induce himself to practise coitus twice, and he was at first made perfectly miserable by his marriage.

In all the cases the abnormal inclination towards individuals of the same sex was congenital (*angeboren*), that is to say, present at the first awakening of the sexual desire, and constant thereafter. Krafft-Ebing very properly places those cases in which the perversion of the sexual sense is only temporary in another category. The gratification of the abnormal desire is effected by masturbation, or by reciprocal onanism. The great proportion remain in a condition of platonic longing, or, at the most, take refuge in self-pollution. Pæderastia is only exceptionally practised, and is loathed by those who do practise it.

Critical Digests and Notices of Books.

On the Minute Structure of the Cerebral Cortex.[1] By Professor Exner.

Professor Exner in this article gives the results of a new method whereby he has lately examined the grey cortex with the special object of tracing the course of the medullated fibres through its various layers. The method employed revealed such a wealth of nerve fibres throughout the cortex that, as he admits, the first glance was quite perplexing to one who had always been accustomed to hardening by chrome salts. A small portion of brain was taken, not over a cubic centimetre in size, and placed in a solution of osmic acid (1 per cent.). The acid is replaced by a fresh quantity after two days, and upon the expiration of five to ten days the preparation is usually found hardened and stained throughout. It is then removed, well washed in water, and then placed for a second or so in alcohol, whereby it becomes more readily imbedded in the oil and wax mass in an ordinary microtome. Sections are cut in the usual way, but, owing to the deep staining, these sections should be extremely fine and should be transferred immediately to glycerine. The preparation may now be placed on a slide, a drop of strong solution of ammonia added, and allowed to remain uncovered for a short time. Finally the specimen is mounted in "water glass" (soluble silicate of soda). The essential part of the process is the application of ammonia, which has the excellent property of converting the neurologia (Ewald's Neuro-keratine) into a swollen, clear, homogeneous mass; and in unstained fresh sections obtained from frozen brain, brings into view a mass of

[1] 'Sitzb. der K. Akad. der Wissensch.' lxxxiii. Band iii. Abth. Febr. 1881.

medullated fibres, whilst the ganglion cells become almost invisible. He describes the appearance presented by fresh sections under treatment by this reagent very fully. Exner has employed the soluble silicate compound as his mounting medium with advantage to a certain point; but he has found, as others have before him,[1] that eventually the mass becomes troubled by a dense crystalloid deposit which spoils the specimen. Having passed in review our already acquired information upon the course of the medullated strands within the cortex, including the statements made upon this subject by Meynert, Kolliker, Arndt, Henle, and Schwalbe, Professor Exner proceeds to detail the results of his own investigations. He adheres to Meynert's classification, and having examined chiefly the central gyri, deals with what Meynert regards as the five laminated type of cortex. In the first layer of "small scattered cortical nuclei," he meets with the superficial medullated tract discovered by Remak, which, whilst apparent in fresh preparations, is not visible in chrome-hardened brain. In the osmic acid sections, however, he is able to confirm the description afforded by Remak, but finds a wealth of structure here which was wholly unrecognised by that observer, and concludes that the whole of Meynert's first layer may be regarded as one of medullated fibres in a supporting connective web. He figures this layer in an accompanying plate, where it is seen to consist of medullated fibres, varying extremely in their diameter as well as in their direction. Some run vertically to the cortex, others horizontally or obliquely, and the smaller fibres have numerous varicosities along their length. The larger nerve fibres are found in the upper half of this layer, and decrease in number and calibre towards the second layer. Besides the medullated fibres disposed parallel to the surface, others are found ascending from the subjacent layers, which bend round so as also to lie parallel with the surface of the cortex; such arched fibres are never of large calibre, but much more frequently of medium size or of smallest diameter. Exner has never seen here the division of a medullated fibre. He here incidentally refers to the fact that occasionally sections from the same spot will

[1] Vide 'Micrographic Dictionary,' 1875, art. "Preservation."

exhibit but a very shallow layer of the stratum of Remak; that sections lose the appearance of this rich fibre formation, and that the staining even disappears in time. I need not emphasise this as a very serious drawback to the merits of the process. Referring to the separation of the nerve structures by the peculiar action of ammonia, he states that the sections increase to one-third their full diameter by swelling, and hence in calculating the distance of the fibres apart from each other, due allowance must be made for this action. In the crucial sulcus and occipital lobes of the dog, these medullated fibres are seen as in man, but are fewer, and still less rich is this formation in the pigeon. In a pigeon's brain, he examined the area defined by Ferrier as electrically excitable,[1] and finds here a notable peculiarity, in that the majority of Remak's fibres here run parallel to the surface of the cortex and are placed one beneath the other in regular series. As time affects the staining, and the medullated fibres become invisible, another element, unseen previously, starts into view. These are fine, widely extended, arched and decussating fibres, which approach "nearest in appearance to the elastic bundles of the choroid." They are found not only throughout the cortex generally, but in the spinal cord. These elastic fibres have been stated to occur here by Professor Stirling in his recent admirable handbook of Practical Histology.[2] In the brain of the new-born infant, Exner finds here no perfect medullated fibres, but still notes a parallel superficial streak containing cells resembling adult ganglion cells, but double the linear diameter of the latter; his estimate being ·014 mm. for the adult and ·030 mm. for the infant. They lie disposed in couples, or more widely scattered, and often resemble in their appearance and grouping the Purkinje elements of the cerebellum! They are, however, more widely scattered, five such cells being found within a distance of 2·9 mm. These cells are provided with processes, one of which, at least, runs invariably towards the medulla. The relationship of these large nerve-cells of the infant to the smaller adult element is still a moot point. In the second layer of "densely crowded small pyramidal

[1] 'Functions of the Brain' (Fig. 40 x.)
[2] 'Text Book of Practical Histology' (Smith Elder and Co.), page 101.

cells," he finds similar medullated fibres, but fewer in number and smaller in calibre than those of any other cortical layer. In the third layer of "great pyramidal cells" these fibres are found for the first time grouped in bundles. Nearer the medulla the fibres are larger, the fasciculi thicker and more numerous, whilst many run obliquely and "tangential" to the surface. Our author next discusses the nature of the so-called layers, or stripes of Vicq D'Azyr and of Baillarger. The former, which is very evident in the cortex of the occiput, can be readily stained by osmic acid, and is seen to consist of medullated fibres, which do not throughout run parallel to the surface. In a portion of cortex from the temporo-sphenoidal lobe, the stripe of Baillarger was most clearly seen 1 mm. distant from the medulla in the deeper part of a sulcus. Many "tangential" fibres were here found passing in this depth of the cortex from the one lateral wall of the sulcus to that of the other. These arcuate fibres, by their presence, also contributed to the deeper tint of this realm, as their medullary elements were well stained by osmic acid. If we imagine the cortex unfolded, we may regard the stripe of Baillarger as a tract of medullated fibres lying parallel to the surface of the cortex. Beyond the above facts, Professor Exner can afford no explanation of the direction and functional relationships of these two medullated tracts, yet he points out the probable relationships of the stripe of Vicq D'Azyr to the visual centre as it lies closely connected with the angular gyrus. In the fourth layer of "small densely congregated irregular cortical cells," the medullated fibres increase rapidly in number towards the medulla, leaving few intervals betwixt them, and run in all possible directions, but most commonly take the "tangential" course. The layer of spindle cells is not alluded to. The article is accompanied by a very excellent plate of the appearances of each layer of the cortex, prepared by the osmic acid method.

BEVAN LEWIS.

Illusions. A Psychological Study. By JAMES SULLY. London, Kegan Paul and Co.

THIS new volume of the International Scientific Series is a welcome variety in this vast mess of intellectual food, this olla podrida of knowledge, to which it adds a distinctly new flavour. And one important quality of the work is that it is strictly scientific so far as it goes, and that on no point does the author more thoroughly elaborate the exposition of his subject than in drawing and defining the sharpest boundary thereof as against the illimitable tracts of philosophy, whereon the humble plodder after real knowledge, whether it be that of so-called common-sense or the more methodical knowledge which ventures to call itself science, may be so readily lost in desert and mirage. According to the philosophy of the Bishop of Cloyne, so hard to believe and still harder to refute, all is illusion. "The great globe itself and all which it inhabit," are not merely like "the baseless fabric of a vision," but actually are such, so that our belief in any kind of knowledge outside the knowledge of the working of one's own individual mind is but one huge illusion. But not so does Mr. Sully read the fairly uniform results of experience, whether it be the vulgar experience of common men or the methodised experience of such men as those who write and those who read the International Series. Let philosophy have its fling and its say, rending knowledge into an impalpable *débris;* psychology, standing firm upon the common experience of myriads of witnesses, declares that there is a reality both of mind and of matter, although the relations of the two are often twisted and misleading.

Quoting Shakspeare, reminds us that the author, though chary of illustration to a degree which renders his pages drier than they need otherwise have been, is not always happy in their selection. At page 3, for instance, after explaining that he uses the term "illusion" in its wide general sense, and not in the restricted sense of "the phenomena of illusion ordinarily investigated by alienists," he remarks that "the play of fancy which leads to a detection of animal or other forms in clouds, is known to be an occupation of the insane, and is rightly made

use of by Shakspeare as a mark of incipient mental aberration in Hamlet." This statement, however, is probably only an illusion of memory, for it is Hamlet who himself applies the test, not to himself, but to Polonius, to see how far the tedious old fool will fool him to the top of his bent. There is not the slightest ground for the supposition that Shakspeare meant to represent Hamlet as thinking the cloud like a whale or a weasel. He used this cloud shape for Polonius, as he used the stops of the recorder for Rosencrantz and Guildenstern to expound his insight into the thoughts of his fellow men, and not as indications of his own mental state, even in the motley disposition which he had chosen to put on.

The author explains that the subject of his treatise, or, to use his own more appropriate term, of his study, is in the more extended and popular sense of the term illusion, as when we commonly speak of a man being under an illusion respecting himself when he has a ridiculously exaggerated view of his own importance, and in a similar way of a person being in a state of illusion with respect to the past, when through faulty memory he pictures it quite otherwise than it is certainly known to have been.

"Taking this view of illusion, we may provisionally define it as any species of error which counterfeits the form of immediate, self-evident, or intuitive knowledge, whether as sense-perception, or otherwise. Whenever a thing is believed on its own evidence and not as a conclusion from something else, and the thing then believed is demonstrably wrong, there is an illusion. The term would thus appear to cover all varieties of error which are not recognised as fallacies or false inferences. If for the present we roughly divide all our knowledge into the two regions of primary or intuitive, and secondary or inferential knowledge, we see that illusion is false or spurious knowledge of the first kind, fallacy false or spurious knowledge of the second kind. At the same time, it is to be remembered that this division is only a very rough one. As will appear in the course of our investigation, the same error may be called either a fallacy or an illusion, according as we are thinking of its original mode of production or of the form which it finally assumes; and a thorough-going psychological analysis of error may discover that these two classes are at bottom very similar."

This passage very candidly opens up a fundamental question, upon the answer to which the psychological value of the work very much depends, namely whether these two regions of primary or intuitive, and of secondary or inferential knowledge do really coexist. Is there such a thing as intuitive knowledge? Is a sensation or an emotion knowledge? Is there not always a flash of inference in all knowledge, even in that which is most primitive?

Certain it is that very many of the illusions described in these pages as distinct from errors of the reasoning processes—that is, as distinct from fallacies—are capable of explanation as errors of inferential knowledge, that is to say, as fallacies. It is candid, indeed, of the author to admit this weakness, but unless he can overcome it, his method necessarily crumbles; and nothing can show the difficulty of his position in this respect better than a consideration of Jeremy Bentham's 'Book of Fallacies,' which contains a good store of examples of what Mr. Sully calls illusions.

This uncertainty in the classification of mental error appears even in the earlier chapters on the most simple illusions, and those one would have thought most removed from the effect of reasoning processes, namely illusions of perception. A fly upon the window-pane taken for a bird in the air, a bandage on the body taken for a weight, the two points of a compass taken for one on parts of the skin where the sensibility is low or slow, are illusions of perception owing to defective definition, which may arise either from lack of attention to the sense-impression, as in the first instance, or from a coarse or indiscriminating sense-impression, as in the last. But surely in all cases there is something of inference. We wrongly infer that the fly on the pane is a bird in the air, because it appears poised in space. We wrongly infer that we are pricked with one instead of with two points, because we feel the sense of pain confined to one spot. To call these things illusions may be right, but to classify such errors as distinct from fallacies is surely a somewhat strained method. And this will, perhaps, appear more strongly if we supplement the simple instances above given from the work, by more complicated ones, one of which has simultaneously occurred to us

and to two friends with us even now. We saw and remarked upon a fine laburnum-tree in full flower; but on our saying how can a laburnum-tree be in flower in September? the tree was more accurately observed, and the apparent yellow racemes of flower were then seen to be but the sere leaves of autumn. Surely inference was busily at work in the whole of the illusion, and in its correction. We inferred from the shape and colour of the tree that it was a laburnum in flower; and then we inferred from the time of the year that it was not a laburnum in flower, and the process, both in the error and its correction, can scarcely be called "unconscious inferences," as in the following passage, wherein this point seems almost yielded, notwithstanding that the scheme of classification must go with it.

"Speaking generally, one may describe an illusion of perception a misinterpretation. The wrong kind of interpretative mental image gets combined with the impression, or if with Helmholtz we regard perception as a process of "unconscious inference," we may say that these illusions involve an unconscious fallacious conclusion. Or, looking at the physical side of the operation, it may be said that the central course taken by the nervous process does not correspond to the external relations of the moment."

The author divides illusions of perception into those which are passive, and those which are active. The passive illusions arise from without, the sense impression being the starting-point, while the active illusions arise from within, from an independent or spontaneous activity of the imagination. The fourth chapter, on passive illusions as determined by the organism, and the fifth chapter, on passive illusions as determined by the environment, are excellent in variety of example and clearness of explanation; and in the sixth, when the author deals with active illusions, or those to which he attributes an internal origin, the questions arising attain a still higher importance. If we rightly follow the explanation given of the transition of illusion into hallucination, it is, that the two states shade one into the other so gradually that it is impossible to draw any sharp line of demarcation between them; hallucination differing from illusion only in the proportion in which external causes are present, the imaginative impulse becoming

at last self-sufficient and independent of any external impression.

The author divides hallucinations into two fairly distinct forms, namely those which have the semblance of an external impression, with the minimum amount of interpretation, and those which counterfeit a completely developed percept. The first he calls rudimentary, the second developed hallucinations. He thinks that those of the rudimentary sort are most frequently due to peripheral disturbance, while he sees reason to believe that "the automatic excitation of the centres would pretty certainly issue in the semblance of some definite familiar variety of sense-impression." To use the language of Baillarger, they are either "psycho-sensoreal or purely psychical," and it is a curious fact sustaining the author's opinions, but not alluded to by him, that what he calls developed hallucinations are not prevented by the loss of the sense-organ. We have known blind men tormented with hallucinations of sight, and deaf men possessed with hallucinations of hearing; and we see lunatics vainly endeavouring to shut out visual hallucinations by closing or covering the eyes, and others as vainly attempting to exclude hallucination voices by stopping the ears. They can thus exclude natural sights and sounds, but not the morbid ones. On the other hand, developed hallucinations may sometimes be referred to the sense-organ, that is to the periphery, as in those cases where one part of the field of vision only is hallucinated.

The author is more speculative than convincing in his exposition of the manner in which hallucinated insanity develops from a state of sane illusion through a border land of subjective sensations.

"The degree of illusion increases in proportion as the imaginative element gains in force relatively to the present impression, till, in the wild illusions of the insane, the amount of actual impression becomes evanescent.

"In these ways, then, the slight, scarcely noticeable, illusions of normal life lead up to the most startling hallucinations of abnormal life. From the two poles of the higher centres of attention and imagination on one side, and the lower regions of nervous action involved in sensation on the other side, issue forces which

may, under certain circumstances, develop into full hallucinatory percepts. Thus closely is healthy attached to morbid mental life. There seems to be no sudden break between our most sober everyday recognitions of familiar objects, and the wildest hallucinations of the demented. As we pass from the former to the latter we find that there is never any abrupt transition, never any addition of perfectly new elements, but only that the old elements go on combining in ever new proportions."

Which is all very fine speculation from the outside writer's point of view, and quite tallies with the greatest poetic authority as to the madman lover and poet being of imagination all compact, &c., but as comparable with true knowledge it is simply "illusion." Lunatics are not the most imaginative of beings, but mostly the dullest of mortals, who, having got hold of a false image, stick to it with stupid persistency. There is not a border land between sane illusion and insane hallucinations, but a clean and clear cleavage, or rather a different stratum, and it is the only pessimist part of Mr. Sully's new work that he can see so little difference essentially between the sane man and the lunatic. It is quite fair also to say that it is the part in which he shows the least amount of knowledge.

The author, in continuation of this subject, propounds a theory, or, as he modestly styles it, an hypothesis, which, so far as we know, is original, and which certainly deserves attention.

"We may, perhaps, express this point of connection between the illusions of normal life and insanity by help of a physiological hypothesis. If the nervous system has been slowly built up, during the course of human history, into its present complex form, it follows that those nervous structures and connections which have to do with the higher intellectual processes, or which represent the larger and more general relations of our experience, have been most recently evolved. Consequently, they would be the least deeply organized, and so the least stable; that is to say, the most liable to be thrown *hors de combat.* This is what happens temporarily in the case of the sane, when the mind is held fast by an illusion. And, in states of insanity, we see the process of nervous dissolution beginning with these same nervous structures, and so taking the reverse order of the process of evolution. And thus, we may say that throughout the mental life of the most sane of us, these higher and more delicately balanced structures are

constantly in danger of being reduced to that state of inefficiency, which in its full manifestation is mental disease."

This is a new illustration of what, in poor Lord George Bentinck, used to be called the stable mind, and it bears very decidedly upon the much-disputed question as to the influence of civilisation upon insanity. We are not aware that the ancient Greeks were peculiarly liable to mental disease, although it is certain that they were no strangers to the higher intellectual processes, and in the insanity of the present day it is by no means an established fact that those members of the community are most liable to the infliction in whom the higher intellectual processes are most developed. Modern statistics of insanity rather go to prove the opposite, seeing that they show the greatest increase in the number of the insane, proportionate to the different classes of the community, to have taken place in the lower and not in the higher classes. It is the pauper lunatic asylums which are filled to overflowing, and which are constantly extending their limits until they have become little cities of the mad; while it certainly has not been proved that really better education and really civilised habits of life have had any influence upon the increase of insanity. But what are "the higher intellectual processes?" Speculation? But what kind of speculation, philosophic or financial? Imagination? But what imagination, the poetic or the spiritualistic? For our own part we verily believe that the best development of civilisation and the highest intellectual processes, so far from being unstable acquisitions tending to early and easy processes of nervous dissolution, are the most powerful of mental antiseptics, and the most wholesome and enduring of mental influences of any kind whatsoever. We do not find our great judges or our great physicians, or even our great poets or statesmen becoming insane; and not an undue proportion even of the little great ones. Moreover, it is quite a mistake to suppose that the higher intellectual processes, depending upon the higher and more delicately balanced structures, are more liable to be reduced to that state of inefficiency which in its full manifestation is mental disease, than are the lower, and, as is assumed, the more stable ones; the fact being that the earliest invasion of mental disease quite as often

attacks in the first place the memory or the emotions, constituents of mind before human history had developed into its present complex form. No doubt the evolution of the race will eventually modify the abnormal mental conditions of the individuals who compose it; but evolution is a slow process, and has scarcely produced any changes which we can at present distinguish, since the time when we first began to inquire about the nature of illusions and hallucinations.

The chapters on dreams are excellent, and open to no criticism. The author has studied the best authorities on the subject, and has also carefully observed the phenomena in himself, so that he knows a great deal about this subject from those direct sources of information, which have evidently been wanting in the matter of insanity. He therefore describes more and speculates less about dreams than he does about the more important subject which precedes it. A curious fact is quoted, showing how the central nervous action of a dream may be projected outwards to the periphery.

"The physiologist Gruithuisen had a dream, in which the principal feature was a violet flame, and which left behind it, *after waking*, for an appreciable duration, a complementary image of a yellow spot."

Some people are said never to dream. Lessing asserted of himself that he did not know what it was to dream. We have met with cases in which insanity was said to have been produced by bad dreams, and we have known of persons who could not be persuaded by good reasons, having been persuaded by bad dreams. But although Sir Thomas Browne thought fit to assert that we are responsible for our dreams, it does not seem very much to matter what we dream, or whether we dream or not.

The suggestion of dreams by slight sense-impressions during sleep, by waking events to which little attention had been paid, and by organic sensations, are well considered; and also the incoherence and the coherence of dreams, and the voluntary attention which we sometimes pay to them. The curious and important phenomenon of after-dreams, or the image of a dream remaining after waking, is referred to. It is well worthy of

more attention than it has received, for it is probably the occasional cause of otherwise inexplicable offences. The author considers the hypnotic condition somewhat briefly. The hypnotized patient, he says, is nearer the condition of waking illusion than is the dreamer, the peculiarities of hypnotism being the concentration of attention in a remarkably narrow field of mental images and ideas; whereas dreams are crowded with continually changing images. Also the hypnotised subject tends to act out his illusions; whereas in dreams the impulses to movement which accompany all percepts are restricted to the stage of faint nascent stirrings of motor activity, which often do not betray themselves externally.

From dream illusions the author passes to quite different matter—the illusions of introspection—and he defines introspection as "the mind's immediate reflective cognition of its own states as such." He admits that all introspection must strictly be considered retrospection, and yet this does not hinder him from drawing a broad distinction between acts of retrospection and acts of memory.

"To begin with, then, an act of introspection, to be complete, clearly involves the apprehension of an internal feeling or idea as something mental and marked off from the region of external experience. This distinct recognition of internal states of mind as such, in opposition to external impressions, is by no means easy, but presupposes a certain degree of intellectual culture, and a measure of the power of abstract attention."

The author remarks upon the tendency in uncultured persons to confuse internal with external experience. External feelings, though they may come in groups, do not fuse into simple wholes as our internal feelings do. Again, external impressions persist so that they can be transfixed by a deliberate act of attention; but not so with the internal region of the mind, the composite consciousness of which never remains perfectly uniform for the shortest conceivable duration. The probability of error in judging of this state is obvious. The intricacies of feelings, their faintness, or their fugitive nature may each or all mislead. The difficulty of attention to these feelings miscalled phenomena, and the still greater difficulty of distinguishing them as they exist from inferences grounded upon them, are

other sources of error. And yet, compared with the illusions of sense-perception, the illusions of introspection are limited. There are no hallucinations, so to say, in the sphere of the inner mental life; and the author believes that, as the power of introspection is a comparatively new acquisition of the human race, as it improves, the amount of error connected with its operation may reasonably be expected to become infinitesimal. This certainly is not a pessimist conviction, but on the whole, we are inclined to think that the more people get into the habit of examining the works of their watches, the worse they will go. And perhaps the author thinks so too, as he has a saving clause as to the calmest and more efficient kind of introspection, and its use in bringing to light what is permanent compared with what is variable in the individual cognition. It may, moreover, be objected to these views that this art of introspection has not been discovered very recently, but that it has long ago been employed not merely by a few cultured experts, seeing that some of the oldest religions and philosophies show it to have been rife among some of the oldest races of men.

To use the old terminology, we have noticed the illusions of the object in those of sense-impression and those of the subject in illusions of introspection. We have now to consider that which it is difficult to classify either as object or subject, namely errors as to the consciousness of others, which the author calls illusions of insight.

"There remains one further mode of cognition which approximates in character to presentative knowledge, and is closely related to external perception. I refer to the commonly called 'intuitive' process by which we apprehend the feelings and thoughts of other minds through the external signs of movement, vocal sound, &c., which makes up expression and language. This kind of knowledge, which is not sufficiently marked off from external perception on the one side and introspection on the other, I venture to call Insight.

"I am well aware that this interpretation of the mental states of others is commonly described as a process of inference involving a conscious reference to our own similar experiences. I willingly grant that it is often so. At the same time, it must be perfectly plain that it is not always so. It is, indeed, doubtful whether in its first stages in early life it is invariably so, for there seem to be

good reasons for attributing to the infant mind a certain degree of instinctive or inherited capability in making out the looks and tones of others. And, however this may be, it is certain that with the progress of life a good part of this interpretation comes to be automatic or unconscious, approximating in character to a sense-perception. To recognize contentment in a placid smile is, one would say, hardly less immediate and intuitive than to recognize the coolness of a stream."

We judge of other persons' feelings not by sense-impression thereof, or by any kind of observation thereof, but by observation of the signs of these feelings—a process of inference, as we venture to think, even in the child; and the author admits that "our insights, like our impressions, though intuitive in form, are obviously determined by previous experience, association, and habit." But the examples he adduces of what he calls the illusions of this intuition, but what we should call the mistakes made in this process of inference, are clearly mistakes as to the interpretation of signs. We infer from a sign, a tone of voice, a word, or a movement, some meaning which it does not really bear, "hastily reading common interpretations into exceptional cases." Wilful deception on the part of the persons observed or unheeded is another source of illusion. Histrionic illusion is another. Desire for sympathy, and the tendency to read in others the feelings by which we ourselves are actuated, is another. And this illusion the author, with subtle discernment, detects in the manner in which we read literature, "approaching an author with a predisposition to read our own habits of thought and sentiment into his words."

Illusions of this nature are so common and so mischievous, that the author concludes his remarks upon them by a kind of apology, and the expression of a hope of the amelioration of our hard lot in this matter.

"In view of this depressing amount of error, our solace must be found in the reflection that this seemingly perfect instrument of intuitive insight is, in reality, like that of introspection, in process of being fashioned. Mutual comprehension has only become necessary since man entered the social state, and this, to judge by the evolutionist's measure of time, is not so long ago.

A mental structure so complex and delicate requires for its development a proportionate degree of exercise, and it is not reasonable to look yet for perfect precision of action. Nevertheless, we may hope that, with the advance of social development, the faculty is continually gaining in precision and certainty. And, indeed, this hope is already assured to us in the fact that the faculty has begun to criticise itself, to distinguish between an erroneous and a true form of its operation. In fact, all that has been here said about illusions of insight has involved the assumption that intellectual culture sharpens the power and makes it less liable to err."

The chapters on illusions of memory are particularly clear, practical, and useful. They might well be studied with profit by every barrister bent on perfecting his system of cross-examination by clearer knowledge of the source of misstatements of fact which are not lies, which we would fain believe is the nature of the great bulk of the disguises of truth which are torn aside in such wholesale fashion in courts of justice. We should have been glad to have condensed this excellent chapter, but we have come to the limits of our space, and must conclude by hearty expressions of approval of this careful and really scientific work in a department of thought and research wherein science is not very much at home. We have had to criticise the classification and the method, or rather the groundwork, of the psychology, seeing inferences where the author sees intuition, and fallacy where he sees illusion; but call the sources of mental error dealt with what you may, the detail of their exposure is acute, scientific, and likely to be most useful, for it will do any man good to study this undogmatic "study" of error, if only to find out some unknown stones of stumbling which may lie in his own way.

JOHN CHARLES BUCKNILL.

Rheumatism: Its Nature, its Pathology, and its Successful Treatment. By T. J. MACLAGAN, M.D. London, Pickering and Co., 1881. 8vo. pp. 328.

DR. MACLAGAN speaks with high authority on the subject of rheumatism, the treatment of which he may be said to have

revolutionised during the last few years. The salicyl compounds which he recommended for the treatment of acute rheumatism in 1876 have steadily advanced in favour since that date, and it is little wonder that they have done so, if, as Dr. Maclagan confidently maintains, under their judicious use the disease lasts as many hours as it formerly did days, or as many days as it formerly did months, pain being abolished and temperature reduced certainly and speedily. The application of these beneficent agents to the relief of a very grievous class of diseases was not the result of any chance discovery or blind groping, but was suggested by an investigation into the causation and pathology of acute febrile ailments and by a careful clinical study of rheumatic complaints, and a theory as to their nature and origin. A theory which has already borne such excellent practical fruit is certainly entitled to respectful consideration, and Dr. Maclagan's book on Rheumatism, which includes a full explication of that theory, will therefore receive general and careful attention, which it deserves, however, altogether apart from the theory, which runs through it with calm persistency. No one can rise from the perusal of this book, whether accepting or rejecting the hypothesis as to the malarial origin of rheumatism, without being impressed with Dr. Maclagan's keenness as an observer, skill as a dialectician, and gifts as a medical author. He writes with singular clearness and precision, has a constant regard to method and proportion, and concentrates his arguments with masterly adroitness. Avoiding all rhetorical adornment and illustrative simplification, he demands the closest attention of his readers, and yet makes his pages attractive by the force of his reasoning and lucidity of his style. Nothing could be more excellent than certain chapters in which he is grappling clinically with disease, and penetrating through obscure symptoms to a rational pathology; as, for example, the chapters on endocarditis or valvulitis, as he is disposed to call it, on pericarditis and myocarditis.

Dr. Maclagan begins with a succinct summary of the symptoms of acute, subacute, and chronic rheumatism; and of the differential diagnosis of acute gout, rheumatoid arthritis, pyæmia, and gonorrheal rheumatism from the two former of these. He

then deals with the duration of rheumatism, and deduces from the statements of a long array of authorities and from his own earlier experience that acute rheumatism has hitherto had a mean duration of several weeks—its duration being measured by the persistency of pain. To this, however, he adds the very satisfactory statement: "In my own practice now, I have come to regard a case of uncomplicated rheumatic fever, in which pain is not quite gone and temperature at the normal, within forty-eight hours of the time at which treatment commences, as an obstinate one." Having argued that rheumatism has its seat in the white fibrous and serous tissues of the motor apparatus, and especially in those portions which are subject to active movement and strain, and that it consists in an inflammation of a specific character, the result of a special poison circulating in the blood, Dr. Maclagan proceeds, very elaborately, to examine the lactic-acid theory of rheumatism, which he effectually demolishes. The next point is to establish his own theory, which is, that rheumatism is malarial in nature, its poison being a miasm which enters the system from without; and in order to do this thoroughly, he enters into a preliminary and very able disquisition on the nature and mode of action of malarial poisons generally. It is impossible for us to follow Dr. Maclagan through this branch of his subject, or to examine critically his thesis that rheumatism is due to a poison which consists of minute organisms. Admitting that the manner in which he applies his miasmatic theory to the explanation and elucidation of the distinctive phenomena of rheumatism is ingenious and sometimes striking, we must add that his arguments cannot at present be regarded as conclusive, and that occasionally he assumes as facts—facts essential to the progress of his arguments—what are only probabilities. The truth is, that the connection of minute organisms with disease has not yet been satisfactorily worked out. We are not justified in saying that the organisms are the cause of the disease, any more than that the disease is the cause of the organisms. All that can be asserted is, that we have before us two natural processes which seem to be inseparably related. Even in the two diseases, anthracoid disease and relapsing fever, in which a definite relation seems to subsist between the development

of the disease and the organism, much fuller information is still necessary, and there certainly remains an unbridged gap between relapsing fever and rheumatism. While overthrowing the lactic-acid theory of rheumatism, Dr. Maclagan has omitted to assail another and more formidable rival of his own miasmatic hypothesis, and that is the nervous theory of rheumatism, supported by Mr. Jonathan Hutchinson. The view that rheumatism is a catarrhal arthritis, consisting in the main of a liability to joint-disease, brought about by exposure to cold and wet through reflex nervous influence, may perhaps afford as plausible an explanation of all the symptoms of rheumatism, as the theory that it depends on a poison which is introduced from without, and finds its nidus in the fibrous tissues of the motor apparatus of the body. When these two theories come to be pitted against each other, as they inevitably must be one of these days, some light may be thrown upon the controversy, we would venture to suggest, by a consideration of the period of maximum prevalence of rheumatic diseases, as revealed by the Registrar-General's Returns, as interpreted by Dr. Mitchell and Mr. Buchan. Should that period be found to correspond with the season of maximum prevalence of diseases of the nervous system, then there would be good ground for believing that there is a neurotic element in rheumatism; but should it be found to correspond with the season of maximum prevalence of malarial or zymotic diseases, then powerful corroboration would be given to Dr. Maclagan's proposition. While consistently advocating his miasmatic theory, Dr. Maclagan does not altogether lose sight of certain nervous relations in rheumatism, for in treating of rheumatism and chorea he says pointedly, "Rheumatism is essentially a disease of the motor apparatus; chorea is essentially a disease of the motor centres. The existence of the rheumatic diathesis implies a liability to disturbance of the motor apparatus. The motor ganglia are an essential part of this apparatus. Those subject to rheumatism are therefore *cæteris paribus* more likely to have susceptible motor centres than those who are not. Thus the rheumatic diathesis predisposes to chorea."

We have already adverted to the admirable character of those portions of Dr. Maclagan's book which deal with rheuma-

tism of the vasculo-motor apparatus, and we might speak in terms of equal commendation of those sections devoted to the treatment of rheumatism and the mode of action of the cinchona and salicyl compounds. This part of the work it is which will be most often consulted and most widely useful; for by virtue of, or in spite of his theory, Dr. Maclagan's treatment of rheumatism is, as evidenced by the large number of cases quoted, singularly successful. His directions are plain and perspicuous, and no difficulty need henceforth be felt by those who wish to give salicin or salicylic acid a fair trial. Our only regret in reading this part of his work is that he has not devoted more space to the brain symptoms which have been produced by the administration of salicylic acid. This remedy has undoubtedly caused delirium, and even insanity when given in large quantities, while in smaller doses it has induced nervous prostration *tinnitus aurium*, a sense of weight and oppression in the head, and a feeling of general misery. Dr. Maclagan, teaching that the salicyl compounds owe their anti-rheumatic effects to their destructive action on the minute organisms which constitute the poison in that disease, mentions the curious effects of salicylic acid on the nervous system in certain cases, but does not offer any explanation of them, which is the more to be regretted, as it seems likely that the deleterious effects of salicylic acid upon the higher nerve centres may yet guide to its employment medicinally in certain morbid states of those centres. Dr. Maclagan points out that salicin never causes delirium.

In dealing with pericarditis, Dr. Maclagan emphasises the important fact that the onset of pericardial inflammation during acute rheumatism is often attended by delirium, which, with stupor and coma, may indeed constitute its most characteristic symptom throughout. From beginning to end there may not be a single subjective symptom of cardiac disturbance, nothing, in short, but the cerebral disturbance. Such cases in which the nervous symptoms predominate are apt to be mistaken for cases of cerebral rheumatism, and Dr. Maclagan quotes a case from Andral, in which "taciturn delirium followed by coma" was found on *post-mortem* examination to have depended on acute pericardial inflammation, marked by a lymphy deposit

on the membrane, there being no appreciable change in the colour or consistence of the brain or spinal cord or their membranes. It is of course desirable to distinguish such cases from others, comparatively rare, in which there is rheumatic inflammation of the fibrous and serous coverings of the cerebrum; but still it seems allowable to designate them cerebral rheumatism, for the nervous symptoms, although coincident with pericarditis, are clearly dependent on cerebral changes. In Andral's case, the patient presented a group of symptoms, viz. pallor of the countenance, incoherence in answering questions, tremor of the lips, bending back of the head, sudden raising of the trunk at intervals, clonic spasms of the facial muscles, and tetanic rigidity of the extremities, which would now be regarded as pointing very definitely to irritation of certain cerebral centres, in which even grave structural changes might have taken place without being appreciable on mere ocular inspection. Whether by reflex action, by the effects of that increased pyrexia which so frequently accompanies the invasion of pericarditis, or by the direct action of a poison on nerve centres constitutionally unstable, it seems that a morbid change is set up in the brain in such cases. This change should not withdraw attention from the pericardial condition which it signalises, but neither should it be overlooked or disregarded in prognosis and treatment. In referring again to cases of this kind, Dr. Maclagan maintains that what determined the prominence of the nervous symptoms in them was not the rheumatic constitution, and not the inflammatory nature of the case, but the special susceptibility of the nervous systems of those in whom that inflammation occurred.

On what appear somewhat insufficient grounds, Dr. Maclagan discards the embolic theory of the connection of chorea and rheumatism of Hughlings-Jackson and Broadbent, and substitutes for it a vague statement as to an association of functional disturbance of the external motor apparatus of the body with functional disturbance of its internal motor centres. The difficulty which Dr. Maclagan experiences in understanding why particles of fibrine detached from the cardiac valves should lodge in the region of the corpora striata, rather than in other portions of the encephalon, and why infarcts should not

contemporaneously occur in the lung, spleen, kidneys, &c., may be readily removed by certain anatomical considerations; and his own suggestions as to a co-existing susceptibility to disturbance in the motor apparatus and motor centres, and as to a rheumatic attack being the mere accidental exciting cause of a choreic paroxysm in persons predisposed to that malady, only remove the difficulty a step farther off, and leave untouched the real problem of the connection of chorea and rheumatism in numerous cases where the chorea is not immediately preceded by an acute attack.

With reference to hyperpyrexia in rheumatism, Dr. Maclagan argues that there is a special thermic centre high up in the cord, controlling and regulating the temperature of the body; that this centre is endowed with heat-producing and heat-inhibiting powers; that it has intimate physiological and anatomical relations with other important centres; that it has connected with it a special set of thermal nerves, distinct from the ordinary nerves of sensation; that these nerves are very freely distributed to the skin; that these nerves are irritated during acute rheumatism by an excess of lactic acid affecting their cutaneous expansions; and that this irritation induces in certain cases, and when of a certain degree, disturbance in the thermic centre.

In noticing Dr. Maclagan's treatise on Rheumatism, we have necessarily accentuated those points on which we differ from him, rather than those on which we are in agreement with him. We trust, however, that, whether agreeing or differing from him, we have fitly recognised the unquestionable merits of his work. The treatise is a truly valuable one, provocative of thought, and likely to conduce in no trifling degree to the alleviation of suffering.

J. Crichton-Browne.

Clinical Cases.

A CASE OF TREPHINING.

BY HERBERT W. PAGE, M.A., F.R.C.S.

ELIZABETH W., aged 19, came to St. Mary's Hospital on November 20, 1880, having been struck on the side of the head by a small conical bullet, accidently discharged from a revolver by a man standing near her. A small scalp wound was found a little above and behind the right frontal eminence, and thence a track led backwards and inwards under the scalp for an inch and a half towards the bullet, which was extracted, after fresh incision upon it, by Mr. Wills, house surgeon. The bullet was found to be flattened, but no obvious damage had been inflicted upon the skull. She refused to stay in the hospital, and again refused on the morning of the 22nd, when she came to have the wound dressed, and when slight elevation of temperature, some swelling about the wound, and complaint of pain over the right side of the head pointed strongly to the advisability of her becoming an in-patient. These symptoms being rather worse on the following day, the 23rd, she at length consented to stay in the hospital. The wound was ordered to be poulticed. The temperature rose that evening to 103° F., and she was slightly delirious; but on the two following days she seems to have improved, for the report states that the temperature fell, the wound looked better, she ate and slept well, and had but little pain. I first saw her on November 26, and advised that the sinus between the two orifices should be laid open, as pus was being pent up under the scalp. In other respects she seemed well.

On November 27 I went to inquire after her at 5 P.M. and learned from the nurse that she "had had four hysterical fits that morning, the last at 2 P.M.; 'hysterical' because they began with crying, and she also cried after she came out of them. Face and limbs twitched, but one side not more than the other. She was certainly unconscious for ten minutes."

I found her drowsy, with a pulse of 120, and a temperature of 105° F. The right side of the face seemed to be a little drawn, and although she squeezed well with the right hand,

some difficulty was found in getting her to squeeze at all with the left. There was no perceptible deviation of the tongue. The pupils were equal and active. A consultation was called, and Mr. Pye and Mr. Pepper met me at 8.30 P.M. The hemiplegia was then decided. There was drawing of the mouth and obliteration of the left naso-labial fold, with an absence of wrinkling of the left side of the face when showing her teeth. P. 112, T. 104·8° F. I accordingly trephined at the spot where the bullet had struck, with the expectation of finding death or inflammation of bone and pus either between the skull and dura mater, or between that membrane and the brain. Although bare over an area of the size of half-a-crown, the bone was found alive and healthy, and immediately underneath it, rather firmly adherent to the dura mater, was a thin clot of blood. The clot having been picked away with a director, the dura mater was seen to be healthy, and the brain pulsated naturally. A probe was passed in every direction between the dura mater and the bone, and in doing so there welled out from the posterior part three or four drops of pus. The probe could not be pushed more than a third of an inch in this backward direction. The operation was performed and the wound dressed by the Listerian method.

On November 28 there was no material change in her general condition, though she expressed herself as feeling better. The note runs: "Pupils equal and active. *Query*, Can she, or can she not, look to the left? Although I can get her to look at my hand held to her right, I cannot do so when it is held to her left. She is, however, very drowsy, though perhaps roused more easily than last night." Ordered—Pulv. hydrarg. c. cret. gr. ij. omn. 6 hor.

Nov. 29th.—Has passed a restless and sleepless night, and complained greatly of pain in her head. The hemiplegia is more marked, the tongue deviates strongly to the left, and she says she has no power to squeeze with the left hand. The left leg is obviously weaker than the right. There is now left internal squint, and this eye cannot be abducted beyond the central line. She has complained much of pain all over the right side of the head, in the occiput, neck and shoulders. Dressing the wound, or the moving of the head which this necessitated, seemed to cause great pain, and she kept calling out that the pain was so bad that she would die. She is perfectly rational, and at once recognised Dr. Broadbent, under whose care she had been in the hospital two years before with a tumour in the left loin. The gauze dressings are to be removed and an ice-bag constantly applied to the head. To take twenty grains of bromide of potassium every four hours, as she has had hardly any sleep.

Nov. 30th.—Slept a little. General condition better. Less pain. Pulse 92. At Dr. Broadbent's suggestion a sixth of a grain of calomel was ordered every hour.

Dec. 1st.—A much quieter night. Asks for food. The left internal strabismus is more marked, and the right eye also, though not squinting inwardly—cannot now be abducted beyond the central line. The eyes look strangely staring, for the lids are wide apart and the pupils are both widely dilated. They react to light. The margins of both optic discs are clearly defined, but the veins are unduly prominent.

Dec. 2nd.—She seems weaker, but is taking plenty of food. Gums a little touched. The appearance of the optic discs has changed since yesterday. The apparent lower margin of the right is somewhat ill-defined; the whole disc is pinkish in hue and the veins are large. The margin of the left disc, which is decidedly paler than the right, is perfectly clear. The veins are large.

Dec. 3rd.—Very weak, but complaining much less of pain. The squint of the left eye is not so marked, and the tongue is protruded straighter. Calomel every two hours.

Dec. 4th and *5th.*—Temperature gradually falling, and decided general improvement.

Dec. 6th.—Looks and is certainly better and stronger. Can grasp pretty firmly with the left hand. The tongue is protruded not nearly so much to the left, she shows her teeth more evenly, and the left naso-labial fold is again distinct. The left internal squint is much less, and she has considerable power to turn the eye to the left. The right eye also can now be moved outwards beyond the middle line. The pupils, equal and active, are still widely dilated, and the eyes are staring as before. There is no change in the left optic disc since the last note, but there are marked changes in the right. The margin is woolly; there is slight swelling, and the vessels are here and there obscured. There are no hæmorrhages. She has hardly any pain, and in all respects seems better. She had a very good night.

Dec. 8th.—No marked change. She has had pain in both legs, and there is slight effusion in both knee-joints. On this and the two following days she continued to improve, and the temperature was normal. The pain in the legs was relieved by fomentation. The difference between the two optic discs remained the same, though if anything the veins of the left were not quite so turgid.

Dec. 11th.—After a quiet night she woke early, and soon passed into a state of coma, which gradually deepened throughout the day. The left pupil became slightly smaller than the right. She died at 4.30 P.M.

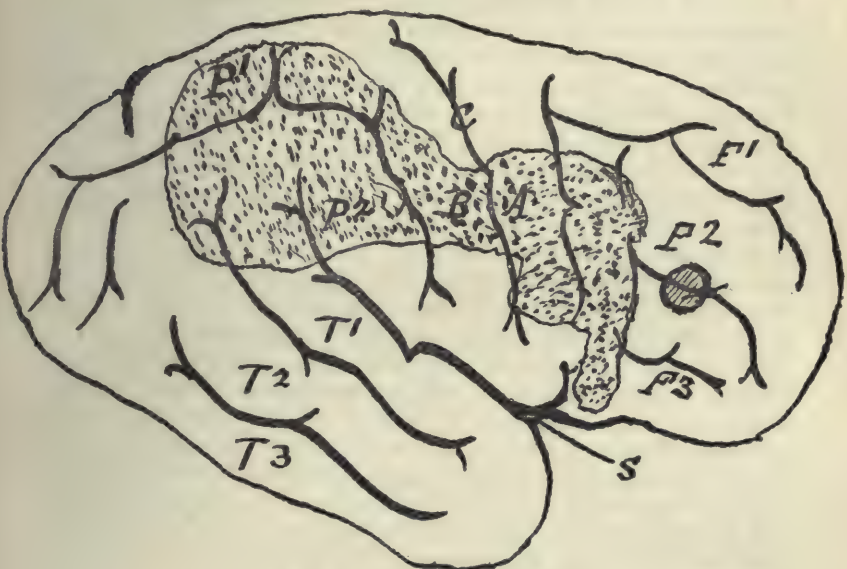
The following is the record of temperature taken at 10 A.M. and 8 P.M.

	Deg. Fahr.	Deg. Fahr.
Nov. 27	98·4	104·8
„ 28	101·6	103·6
„ 29	101·4	102·2
„ 30	102·6	102·0
Dec. 1	102·0	101·8
„ 2	101·6	101·4
„ 3	101·2	100·6
„ 4	99·8	101·8
„ 5	100·8	99·2
„ 6	99·8	99·0
„ 7	100·0	99·6
„ 8	98·8	98·8
„ 9	99·4	99·0
„ 10	98·4	98·8
„ 11	98·8	101·2 at 2.30 P.M.

The autopsy was made by Mr. Pepper, whom I must thank for the following record and diagram:—A crucial incision has been made through the scalp midway between the right orbit and the sagittal suture, and behind a line drawn perpendicularly up from the outer angle of the orbit; the posterior border of the circular trephine wound is $\frac{1}{8}$ inch in front of the coronal suture. The dura mater is uninjured either by the operation or by subsequent sloughing. The bone round the wound is quite healthy. The trephine aperture in the bone is filled with granulations adherent to the dura mater. The aperture is separated from the abscess to be described by a margin of $\frac{1}{3}$ inch on the surface of the brain where the dura mater was adherent to the pia mater. A section of the brain at the site of operation shows the brain substance to be disintegrated for $\frac{1}{4}$ inch beneath the pia mater. On the surface of the brain is found a collection of pus, perhaps ʒij in quantity, and also a layer of lymph about $\frac{1}{20}$ to $\frac{1}{12}$ inch in thickness, which separated readily from the pia mater, like the false membrane of croup, leaving the convolutions considerably depressed, but not dipping between the sulci. The membranous exudation, or abscess, is on the right hemisphere, and consists of two parts joined by an hour-glass construction, which is placed over the fissure of Rolando. The anterior portion is quadrilateral in form, and measures $1\frac{1}{4}$ inch from before backwards, and $1\frac{1}{2}$ inch from within outwards. Its upper margin is $1\frac{1}{4}$ inch from the median longitudinal fissure. From the anterior and inferior cornu a process runs down on the inferior frontal convolution as far as the anterior limb of the Sylvian fissure. This anterior portion of the abscess occupies the posterior part of the middle

and inferior frontal convolutions, and the lower part of the ascending frontal convolution for the same depth, but does not encroach on the connecting convolution between the ascending frontal and ascending parietal convolutions. The posterior portion reaches almost to the longitudinal fissure, and is irregularly oval in shape. Its longest diameter extends from the constriction backwards and measures $2\frac{3}{4}$ inches, and transversely $1\frac{3}{4}$ inch. It covers nearly half the ascending parietal convolution, stopping short about $\frac{3}{4}$ inch from the posterior limb of the Sylvian fissure, the parietal lobule, the angular and more than the upper half of the supra-marginal convolution (*vide* shaded area in diagram). There is no disease at the base of the brain, of the spinal cord, or its meninges. There is no thrombosis of cerebral vessels.

An examination of the optic discs showed that the right was double the size of the left, and that the vessels were obscured from the centre of the disc to the periphery. The left optic disc was apparently normal.



C. Fissure of Rolando.
F^1. Superior frontal convolution.
F^2. Middle „ „
F^3. Inferior „ „
A. Ascending „ „
B. Ascending parietal „
P^1. Parietal lobule.
P^2. Supra-marginal convolution.
T^1. Superior temporo-sphenoidal.
T^2. Middle „ „
T^3. Inferior „ „
S. Sylvian fissure.
Site of trephining, just below F^2.

The course of this case presented somewhat unusual features. The nature of the accident, and the early onset of hemiplegia led to the belief, which the example of other cases of like injury and with like symptoms seemed to justify, that there was in all probability the following sequence of events:—contusion, inflammation, or death of bone, the formation of pus between it and the brain, and subsequently purulent inflammation of one of the membranes spreading over the hemisphere to the base of the brain and there involving the special nerves which became paralysed. There actually was present a small extravasation of blood between the bone and dura mater at the spot where the skull was struck, and separated from this hæmorrhage—which might in itself have been harmless—was the localised arachnitis, or abscess, which was limited to a comparatively small surface of the hemisphere, and which compressed the convolutions over which it lay. Had the real character of the lesion been known, it seems possible that good might have accrued from another opening by the trephine.

The paralysis of the ocular muscles deserves remark. The first to be noticed was that of the left external rectus, causing internal strabismus, and inability to turn the left eye outwards. This was soon followed by inability to turn the right eye outwards, but notwithstanding this paralysis of the right external rectus, there was no internal strabismus on the right side, and it was only when the patient was asked to look to the right that there was any evidence of defective power in the movements of the right eye. The paralysis on the right side—the side of the lesion—was therefore not so extreme as that on the opposite side, or side of the hemiplegia. It was this seeming involvement of both sixth nerves which led in large measure to a belief in the existence of purulent meningitis spreading to the base, a conclusion supported also by the pain in the back of the head and neck, and by a tendency towards opisthotonos when the pain was most severe. There was never any conjugate deviation of the eyes and head, although the first note—on which indeed her drowsiness threw doubt—that she seemed unable to look to the left, rather pointed to impairment of power over the associated movements of the left external and the right internal recti muscles. Noticeable also were the wide dilatation of the pupils and opening of the eyes.[1]

Optic neuritis, or the appearances indicating it, developed on the side of the brain lesion, while the turgidity of veins, originally the same in both eyes, diminished somewhat in the

[1] Where this phenomenon has been induced by experimental irritation it seems always to have been accompanied by turning of the head and eyes towards the opposite side. *Vide* 'Functions of the Brain,' pp. 143, 151, 152.

left, or opposite eye, during the last three days of life. If the fulness of the retinal veins were due to obstructive pressure,—and if that be an immediate cause of optic neuritis—there was no apparent reason why the neuritis should have affected one side only, for the earlier appearances of the two sides were precisely the same. Had the girl lived longer it is of course possible that the optic neuritis might have become double, but it is perhaps not merely a fortuitous circumstance that optic neuritis developed only on the side of the lesion. There was no trace of basic meningitis; and it is unfortunate that the length of time which elapsed before the *post-mortem* could be made precluded any microscopic examination of the optic nerves.

CASE ILLUSTRATING CEREBRAL LOCALISATION.

BY DR. ALEXANDER ROBERTSON,

Town's Hospital and Asylum, Glasgow,

and

DR. DAVID FOULIS,

Pathologist to the Royal Infirmary, Glasgow.

MICHAEL H., age 23, was the eldest of a family of nine, all of whom were healthy and vigorous except himself. At Michael's birth his mother's labour was severe and protracted, and the forceps were used to aid delivery. His father thinks that his head was injured by the instrument. As an infant he never seemed able to move the right arm or leg, and a squint of the right eye was likewise observed by the parents. "When he was about eighteen months old," his father remarked, "we were thoroughly convinced that Michael was palsied on the right side, from his head to the point of his toes." In the course of his boyhood he acquired so much power in the leg that he was able to walk a little, but his hand and arm remained useless. He never learned to speak more than to say "aye" and "no." Notwithstanding these defects, he showed some intelligence, and tried to join with other children in their games. When about twelve years old he had his first epileptic fit, and this disease persisted till he died. At first he had one or two seizures a week, but afterwards they became more frequent. All his life he was uncleanly in his habits, never seeming to have full power over his bladder and bowels. He was an inmate of the Town's Hospital for about a year before his death, which occurred on the 6th of December, 1880. His condition, while under observation, was as follows, after a series of severe bilateral convulsive seizures :—

The right upper extremity was wasted and powerless; the forearm was flexed upon the arm, and the hand upon the forearm; the fingers were contracted upon the palm, and could

not be straightened. He was able to walk, but had the paralytic drag of the right foot in a well-marked form. There was no obvious defect on the left side. His stock of language still consisted of "aye" and "no," which he appeared to use intelligently. There was very little droop of the mouth on the affected side. So far as could be ascertained by pinching, there was no defect of sensibility in the palsied limbs. He generally smiled in a silly manner when addressed, and was childish and facile. He was certainly neither blind nor deaf, but to what extent the senses of sight and hearing were defective, if they were so at all, could not be determined.

Description of the Brain by Dr. Foulis.—A number of the convolutions have a peculiar shrunken and wrinkled appearance, as if they were divided by a network of wrinkles into smaller convolutions about an eighth of an inch in width.

On the left side the wasted convolutions are:—The lower third of the ascending frontal, and lower half of the ascending parietal, and a small part of the posterior end of the 2nd and 3rd frontal; thence the wasting extends upwards and backwards into the angular gyrus, the whole of which is involved, as well as the superior temporo-sphenoidal convolution. From the angular gyrus the wasting extends backwards in a narrow strip to the longitudinal sinus.

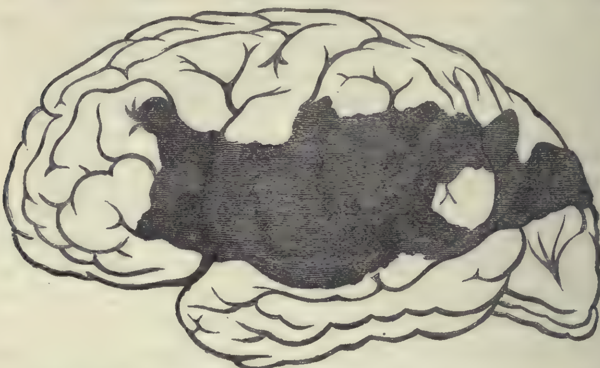
On the right side the wasting involves chiefly the angular gyrus, the supramarginal, and the posterior half of the first temporo-sphenoidal, and slightly the lowest part of the ascending parietal convolutions; it also extends backwards in a strip which runs across the interparietal fissure to the region of the cuneus.

A microscopical examination of the brain-tissue was made; but, owing to the long time during which the brain had been kept in dilute spirit, the cell changes had become obscured, and nothing definite can therefore be said about them.[1]

Remarks by Dr. Robertson.—This case gives both positive and negative support to the prevailing views respecting the localisation of motor function in the cortical substance of the cerebrum. Thus, the palsy being on the right side, the left ascending frontal and parietal convolutions were those in-

[1] The brain was shown at meetings of the Pathological Society of Glasgow and of the Medico-Psychological Association. Besides the changes described by Dr. Foulis, it was perfectly obvious, when the brain was removed from the skull, that the left side of the pons varolii and medulla oblongata was smaller than the right —probably a fourth or a fifth less—and this was pointed out to the former of these meetings. The base of the brain was somewhat flattened by pressure when Dr. Foulis examined it, and he therefore refrained from describing the morbid alteration in the pons and medulla. It is further to be observed that no appreciable difference was observable between the corresponding ganglia on the floor of the lateral ventricles.—A. ROBERTSON.

volved. But, more particularly, the loss of muscular power was much greater in the right arm than in the right leg, while the main defect in these convolutions was at their *lower* part. So also with speech. The operculum and back part of the



Left Hemisphere.—Lesion is mainly behind the ascending median convolutions.

inferior frontal convolution were seriously affected, and articulate language was in almost complete abeyance.

Turning now to the right hemisphere, the main defect was behind the median convolutions, and was thus posterior to the motor region: in further accordance with the views referred to there was no appreciable palsy of the left extremities. This observation must, however, be qualified by the fact that the

Right Hemisphere.—Lesion seriously involves the ascending median convolutions.

lowest part of the ascending parietal convolution was implicated; but the degree in which it was affected was slight,

so that it seemed scarcely likely that the coarser and more general of the movements over which that convolution is believed to preside would suffer thereby. Such a lesion might have been expected to damage or destroy the power of executing the finer and more delicate manipulations, had it existed, but, as the cerebral defect probably began at birth, this power could not have been acquired.

The wasting in the left side of the pons and medulla, with the seeming normal development of the corpus striatum, regarded in relation to the lesions in the motor convolutions, lends some corroboration to the view that there are fibres which ascend directly from the cord to these convolutions, passing by that great ganglion in their upward course.

The general intellectual deficiency did not permit an accurate estimate to be made of the state of the special senses, so that no inferences can be drawn in respect to the lesions in the reputed centres of sight and hearing.

1. CASE OF ELEPHANTIASIS GRÆCORUM.
2. CASE OF ABSCESS OF THE BRAIN.

BY WALTER RIVINGTON, F.R.C.S., ETC.

CASE I.—*Elephantiasis Græcorum. Anæsthetic form in a Boy nine years of age—Amputation of Forearm—Recovery—Remarks.*

ALEXANDER McLENNAN S—N, half-caste, age 9, height 3 feet 11 inches, born in Demerara (George Town), had always enjoyed good health. His parents were—father a Scotchman, mother an Indian who could speak English. His uncle took charge of him, and he learnt to speak English very well. He left Demerara for London seventeen months before admission to the Hospital in June 1873. His first statement to the dresser was that he was in perfect health, and remained so till January 1874, when he took measles. When he was becoming convalescent from the measles, his right hand began to swell. It was not painful, but he gradually lost the use of his hand. The fingers became flexed on the palms, and the extensors lost their power. The skin of the fingers then began to ulcerate over the knuckles, and some brown patches appeared on the skin. There was no history of skin-disease in the family. After the measles, he attended the out-patient department of the German Hospital, under Dr. Burger, who ordered cod-liver oil and warm bathing. Not getting better, he was advised to go into a general hospital, and he became an inmate of the London Hospital in November 1874. On being questioned by Mr. Rivington, the boy admitted that the hand was bad and that the patches appeared prior to the measles. They came on in the winter before Christmas 1873. He had a large dark brown patch, involving the right eyebrow and upper lid, and extending on to the forehead for nearly an inch, being larger than a penny-piece. There was no corresponding patch on the left side. There were two small patches, not so large as threepenny-pieces, over the right malar bone, and two patches, about the size of a sixpence, outside the mouth on the right side. There were two patches on the chin, about equal to

three penny-pieces. There was a patch in front of the left ear equal to a halfpenny, and one, the size of a penny, over the body of the lower jaw on the left side, an inch in front of the angle, and extending under the jaw into the neck. There were five or six other small patches on the left cheek. The skin was naturally of an olive colour, but the patches were of a reddish-brown, scarcely raised above the level of the skin, not tubercular, covered in parts with some scales of epidermis. There was a patch on the lobule of each ear, the left being the larger.

On the left upper limb there were patches chiefly on the dorsal surface of the forearm and one on the palm. The soft parts of the index had swollen, and the skin was broken and discharging blood. On testing the patches with a pin, they were found to be by no means destitute of sensation, but sensation was less acute than on the healthy skin. The patches on the right arm were not symmetrical with those on the left side; the largest patch, with the exception of one on the dorsum of the hand, was on the outer side of the arm above the external condyle. He had patches on the buttocks, on the outer side and anterior surface of thighs, on the legs and feet. The calves were nearly free. He had sores on the feet like those on the hand, but these healed. The stomach was large and prominent, and the upper part of the chest was flat.

The right upper limb was affected with wrist-drop from paralysis of the muscles supplied by the musculo-spiral nerve. The muscles supplied by the median were active. The fingers could not be extended. All the fingers were swollen, the third digit most of all, and there was ulceration over the first phalangeal articulation. There was diminished sensation over the dorsum, which was discoloured, and constituted a large anæsthetic patch. A pin run into the middle finger elicited no sign of pain. The ends of the fingers were acuminated; the nails were very convex on their dorsal aspect, and much incurvated, like claws. The musculo-spiral nerve was much enlarged above the elbow-joint, and could be traced in its enlarged state round the humerus. The median nerve was also readily felt to be enlarged by the side of the brachial artery. The ulnar nerve could be traced in a swollen state from the axilla to the back of the internal condyle. The enlargement of the nerves appeared to begin in the arm just below the axilla. There was an elastic, and apparently fluctuating swelling below the head of the radius and outer side of the forearm.

On the 11th of November, 1874, Mr. Rivington amputated the right forearm at about the junction of the middle and lower

thirds, skin and fascia being dissected up for the flaps, and the muscles being divided circularly. The bones of the forearm were relatively small. Ligatures were applied to the arteries. The patient made a good recovery, and the stump healed well. The highest temperature was on the 12th, 103·6.

After recovery from the operation, the patient was sent to school. He came under Mr. Rivington's care again for a short time about six months later. The general disease had not made any further progress. Since then he has not been seen or heard of.

The amputated part was delivered to Mr. Joseph Needham for microscopic examination and report. Mr. Needham's careful report runs thus:—

"The following portions of the tissues of the hand were submitted to examination, viz., median and ulnar nerves; integument from dorsum of hand.

Ulnar Nerve.—The disease-process may be easily traced in transverse sections of this nerve. In some places the nerve fibres are nearly healthy, but are separated by very small cells and nuclei. Some of the cells are fusiform, but the majority are spheroidal and very small. In other places the nerves are with difficulty seen, the medullary sheath and often the axis cylinder having disappeared, and their situation being occupied by large numbers of the before-mentioned cells. Again, in other nerve bundles the cells have disappeared in the centre, and a finely granular mass occupies their place. This change commences in the centre of bundle of nerves, and nowhere is it observed to extend to the circumference. The blood-vessels are larger than natural, and are surrounded by cells identical with those found in the nerve-trunks. It appears as if the morbid process commenced by an infiltration of the tissues by colourless corpuscles, followed by multiplication of these cells, and also of the connective tissue corpuscles by division. The origin of the small fusiform cells appears to have been the connective tissue or neurilemma.

Median Nerve.—The nerve is in size about four times its normal diameter. The morbid process is far advanced in this nerve. In transverse sections the margin of each bundle is indicated by a host of small corpuscles, agreeing with those already mentioned, whilst the interior appears constructed of a finely granular material. No trace of nerve fibres can be detected in this sclerosed mass. The sheath of the nerve contains here and there patches composed of masses of small cells, giving rise to an appearance similar to that of a microscopic abscess.

Skin.—Similar changes can be easily traced in the skin.

Wherever the skin has a large number of blood-vessels demanded by its peculiarity of construction, as, for example, around sudoriparous, sebaceous and hair follicles, between the connective tissue bands of the corium multitudes of small cells are distinctly visible, forming large tracts and enclosing the structures already mentioned. The vessels can be readily traced, on account of their position being indicated by the number of corpuscles surrounding them. On studying the integument, one is impressed with the belief that the connective tissue has very little to do with the process, the structures formed of this material possessing for the most part a normal appearance."

CASE II.—*Abscess of Brain, following an injury to the skull—Successful trephining, and evacuation of abscess. Reformation of abscess—Evacuation—Recovery.*

(The notes of the case were taken by Mr. Pound, the dresser, who brought the case to Mr. Rivington.)

George M., 31. Came under Mr. Rivington's care at the London Hospital on the 15th of October, 1879. He was a letter-carrier from Derby, and his previous history was of great interest. On the 28th of January, 1876, the patient was passing under some scaffolding, when a file fell on his head, the pointed end striking him perpendicularly; he was wearing an ordinary postman's cap at the time, and the file passed through it. He was seen by a surgeon immediately afterwards, and it was then found that he had sustained apparently a very trifling injury. There was a small wound situated over the posterior superior angle of the right parietal bone, penetrating the scalp, but the bone did not appear to be injured, and the patient seemed none the worse for the accident. Three days afterwards he began to have pain in his head, and he took to his bed. The pain became more and more violent, but there was no loss of consciousness. Evaporating lotions were applied without benefit. He was taken to the Derby Infirmary, where hot poultices were ordered. He rapidly became worse. The pain increased in severity: he became delirious, and there was slight left hemiplegia. On the 24th of February he was trephined—a small piece of bone about the size of a fourpenny-piece being removed by the gouge forceps. A small puncture was then made through the dura mater, when a large quantity of pus escaped, coming through the opening in jets. He was perfectly free from pain on recovering from the effects of the chloroform, and got rapidly better, so that he was discharged

from the Infirmary, cured, on the 27th of March, after which he was able to resume his duties as letter-carrier.

On the 28th of December of the same year he had slight convulsions, which increased in intensity and frequency, and then the left side became paralysed. The old wound was reopened on the 30th of December, letting out a little blood-stained serum. It then appeared that the cicatricial tissue which was filling the aperture had caused the mischief, as he was very soon able to use his left arm and leg, and in February resumed his duties, being then quite well and strong; so well, indeed, that he bought a bicycle, and soon became a good rider. On the 18th of July he was suddenly seized with violent convulsions at the Post-office; previously to this, however, he had complained for a few days of pain in the head, and failing in his left leg. The convulsions became very frequent, and the wound was again reopened on the 22nd of the same month, but this time, seemingly, without any good result, or any relief to the patient. A small piece of dead bone was removed at this time. He was now so ill, that the surgeon did not think he would live many hours. He was carefully nursed, poultices were constantly applied to his head, and he slowly improved. There was a good deal of discharge from the wound, and he complained of severe pain in the paralysed arm and leg. His speech has been thick since the convulsions on the 18th of July, 1879. He has not had any convulsions since the last operation. Whilst in the fits he several times bit his tongue and lost some of his teeth. His sight has lately become affected, for he told his father he should want a pair of spectacles. His father thinks there is no difference in his hearing. He has never passed urine or fæces involuntarily. The last time he did any work was before the operation of the 2nd of July, 1879.

On admission into the London Hospital, a swelling was found with a cicatrix over the posterior superior angle of the right parietal bone; it was of firm consistence, and apparently was composed of dense cicatricial tissue. The patient could not walk without assistance, and even then appeared to drag his legs after him and knock his knees together. He kept his left arm bent across his chest, and did not attempt to move it; when asked to take hold of anything with it he did so, but without much power. He talked thickly, could whistle with an effort, shut his teeth together evenly, and put his tongue out straight. The right naso-labial fold was much better marked than the left. He could close both eyes firmly. He was very restless when put to bed, and was obliged to have an attendant. He answered questions correctly, but was slightly

delirious, and had rather strange ideas about things, e.g. he thought he should be well enough to return to Derby the next day, and that he should be able to go on his duty as a letter-carrier on the following day. He took his food well, and said he had a good appetite. Finding an elastic and apparently fluctuating swelling beneath the old cicatrix, and taking into consideration the previous history and the symptoms of pressure upon the brain, Mr. Rivington diagnosed an abscess in the brain. The wound was reopened, and a small piece of dead bone was found and removed. The comparative mildness of the symptoms was attributed to the fact that the hole in the skull allowed the abscess to bulge outwardly and diminished the pressure on the brain. It was Mr. Rivington's intention to make a deeper exploration, and with that view he showed the case to Mr. Curling and Mr. Hutchinson, who happened to be in the hospital. The paralysis being only partial, they both advised waiting to see if the symptoms deepened. On the 23rd of October Dr. Hughlings-Jackson saw the patient, and examined him with the ophthalmoscope. There was optic neuritis in both eyes. At this time there was hemiplegia on the left side, the leg suffering very much more than the arm; he could not stand, but he could move his fingers. The movements of the shoulder were affected. A day or two later the swelling was observed to be enlarged, and pus was seen issuing in jets from a hole in the top of the head. During the night a very large quantity of pus escaped into the bed. The amount was estimated at about half a pint. After the escape the patient became better in every respect. He recovered the power of walking, his speech was not so thick, and he moved his left arm and fingers freely. He also improved in his mental condition. When Mr. Rivington saw him, he enlarged the external opening, and found that a director would sink by its own weight quite 2 inches towards the centre of the brain before meeting with any resistance, thus demonstrating the existence of an abscess-cavity in the interior of the hemisphere.

On October the 29th, after the escape of the pus, Mr. Couper examined the eye, and reported as follows:—

"There is considerable swelling of both discs; it amounts, by direct ophthalmoscopic measurement, to a hypermetropia of one-eighth in the right eye and one-sixth in the left eye. The right disc is thus greatly more swollen and prominent than the left. There is much increase of connective-tissue in the optic-nerve fibre layer of the disc, and the reticulations of this tissue are distinctly visible by direct examination. The nerve-substance of the disc is thus rendered opaque, and the opacity

extends some little way into the retina, concealing the choroidal boundary of the disc in each eye. There are also apparent interruptions of the vessels as they dip behind the nerve-fibre bundles. The veins of the disc and retina are varicose, but they are not greatly distended. Some red streaks in the discs, looking like hæmorrhages, are really portions of vessels partly concealed by the opaque and altered nerve-structures."

It is interesting to note that the abscess was in the neighbourhood of those convolutions which have been shown experimentally to be concerned in presiding over the movements of the limbs. After the bursting of the abscess, an event which saved the surgeon the necessity of an incision, the patient made rapid progress. The loss of power in the limbs on the left side was reduced to weakness, and the weakness gradually passed away, the optic discs cleared, and the sight correspondingly improved. The discharge diminished day by day, and the patient was shown to the members of the Hunterian Society. Ophthalmoscopic examination of the eyes by Mr. Couper on the 10th of January, 1880, yielded the following results:—"Right disc, not swollen; choroidal margin, indistinct, nerve-substance being greyish-red and not translucent; veins tortuous, and slightly larger than normal, their sheaths being distinctly thickened, those of the arteries not markedly so. The course of the optic nerve-fibre bundles, as they pass from disc to retina, is clearly seen of a radial fibrillar appearance; no hæmorrhages; some greyish change in retina around macula; left disc differs from right chiefly in its choroidal margin, being slightly obscured by grayish connective tissue change in nerve substance of disc; veins, slightly tortuous; sheaths slightly thickened; arteries normal; no swelling."

The progress towards recovery was interrupted at the end of November, when the discharge from the wound greatly increased, and the patient suffered from headache, and was obliged to keep his bed. On December the 1st he had a fit. His own account of the commencement of the attack was this: "I felt it coming on by a convulsive movement of my left fingers; they were drawn up and closed. These movements extended to all the muscles of my arm. I was put to bed, and I do not know what happened for about 2 minutes. When I came to, I was as well as ever." The patient was unconscious for 2 minutes, and the fit lasted 15 minutes. He was convulsed on both sides equally, and spluttered a great deal. Whilst unconscious, he was affected with complete paralysis of his left side; his eyes were turned up; there was no blueness of the lips; he did not bite his tongue. On December the 15th the

wound had closed, and the patient seemed to be quite well. On January the 6th, 1880, he was in good health, and soon afterwards left the hospital at his own request to return to Derby.

The rarity of recovery after trephining for the evacuation of an abscess within the cranium renders this case worthy of record.

Abstracts of British and Foreign Journals.

The Nervous Mechanism of Respiration.—ROSENTHAL, to whom we are indebted for a great part of what we know respecting the nervous mechanism of respiration, finds himself compelled, twenty years after the publication of his investigations, to review the accounts of other experiments that have appeared from time to time during this period, and to repeat his own observations with various modifications. The results of this undertaking are recorded in two elaborate papers in Du Bois-Reymond's *Archiv für Physiologie* (Suppl.-Band, 1880, s. 34; and Heft I. and II., 1881, s. 39). The conclusions which are reached need alone concern us in this place; but the historical sketch of the many elaborate investigations of the physiology of respiration may be recommended as especially worthy of perusal in the original. Rosenthal's summing-up differs remarkably little from his conclusions of 1860. They are as follows:—

1. The vagus contains fibres, which probably arise in the lungs, and which produce, on being faradised, acceleration with weakening of the respiratory movements. If the stimulation of these fibres be increased, the normal respiratory movements pass into moderate arrest in inspiration. These fibres are to be called "regulating fibres."

2. The superior laryngeal nerve contains fibres, which produce, on being faradised, slowing and deepening of the respiratory movements. If the stimulation of these fibres be increased, the respiratory movements are completely arrested. These fibres are to be called "inhibitory nerves" of the respiratory centre, and to be considered analogous with the inhibitory nerves of the heart.

3. The inferior laryngeal nerve contains fibres, which produce, on being faradised, respiratory arrest in expiration. This effect, however, cannot be demonstrated unless the faradisation be powerful, and is not seen in animals that have been narcotised or deprived of the cerebrum; and it must not accordingly be considered as of equivalent importance to the results under (2). More

probably these inferior laryngeal fibres are to be regarded as ordinary sensory nerves, which influence the respiratory movements mediately, like all nerves of the same class.

4. Comparatively large doses of chloral hydrate (0·3 gram. injected into the jugular vein) completely paralyse the regulating fibres of the vagus, but do not affect the action of the inhibitory fibres.

Whilst the relation of the vagus and its branches to the respiratory centre thus continues to attract attention, the evidence increases that our ideas of the *respiratory centre* as a minute spot in the medulla oblongata must be greatly modified. We have already had occasion to refer to comparatively recent observations on this point (*see* 'BRAIN,' Vol. III. p. 263). Langendorff of Königsberg, in the first place, appears to have succeeded in extending the limits of the respiratory centre downwards into the spinal cord, and Christiani upwards into the third ventricle. Experimenting chiefly upon newly-born animals, and with the excitability of the cord increased by strychnia, Langendorff has more recently (Du Bois-Reymond's *Archiv*, 1880, s. 518) found that spontaneous respiratory movements continue for a quarter-of-an-hour after decapitation; that these movements are not only rhythmical but co-ordinated; that irritation of sensory nerves produces the same effects upon the respiration in these animals as in normal animals; and that the activity of these spinal respiratory centres stands in a certain relation to the gaseous constitution of the blood. Thus fresh proof is afforded that there are true spinal centres of respiration, which act both reflexly and automatically. The question next arises, What then is the "respiratory centre" in the medulla? Langendorff suggests that it is a *regulating* centre, with an action which is chiefly inhibitory; and it is this function that explains the effect of puncture or section of the medulla in causing instantaneous respiratory arrest.

In a later communication to the same journal (Heft I. and II., 1881, s. 78), Langendorff describes the results of further investigations of the relations of the several parts of this central respiratory apparatus; starting from the observation which he made in 1879 (*see* 'BRAIN,' *ut supra*), that median section of the medulla, followed by division of one or both vagi, leads to asynchronism of contraction of the two halves of the diaphragm, that is, that median section of the medulla destroys a nervous connection which co-ordinates the action of two respiratory centres, one on either side of the middle line. In accordance with the conclusions reached in the preceding series of experiments, described above, this result

might be otherwise stated as a proof that a connection exists in the medulla between the *regulating* respiratory centres of the two sides. Assuming the existence of this connection, Langendorff constructs a hypothetical arrangement of the central respiratory apparatus as follows:—A nerve-bundle, arising from the nuclei of the vagus and trigeminus of, say, the right side, passes down, partly to the nucleus of the right phrenic and partly to that of the left. A decussation thus occurs somewhat resembling that in the optic chiasm; and this is the connection which is severed by the median incision. After such median section, any interference with the vagus or trigeminus of one side—say the right—will cause a disturbance of the respiratory action of the diaphragm on the same side only, not on the left, and thus asynchronism in the movements of the chest. Interference with both vagi will have a similarly disturbing effect, because it is now recognised to be almost impossible in such experiments to disturb the two vagi equally. It is the existence of the median connection between the two centres, or, more correctly, the existence of a respiratory chiasm, that prevents such acts as coughing, sneezing, &c., from being unilateral only, and corresponding in their situation with the seat of the irritant (*e.g.* disease of one lung) which excites them. Is it possible that disease of the medulla ever leads to asynchronism of the respiratory movements, and to "unilateral cough"?

An interesting extension of our knowledge of the afferent nerves of the respiratory apparatus has just been made by Dr. John Campbell Graham, working under Pflüger (Pflüger's *Archiv*, xxv. s. 379). This observer finds that in rabbits the trunk of the splanchnic nerve below the diaphragm possesses an expiratory or inhibitory action upon the respiratory centre, faradisation causing relaxation of the diaphragm and contraction of the abdominal muscles. The route of transit of impressions from the abdominal viscera to the centre or centres of respiration appears thus to be demonstrated. By further experiments, Dr. Graham was able to trace the fibres from the splanchnic into the dorsal portion of the cord and thence into the medulla.

The Heart.—Trophic Nerves of the Heart.—The influence of the vagi upon the nutrition of the heart, and the effect upon the cardiac musculature of section of these nerves in the neck, are subjects which have been fully discussed in previous *Reports*. (*See* 'Brain,' Vol. I. p. 581, and Vol. II. p. 436). In a lengthy paper in the ***Zeitschrift f. Klin. Medicin***, iii. s. 317, Wassilieff of St. Peters-

burg, gives an account of several series of experiments bearing on the same points. Stated briefly, the results of these investigations entirely confirm those of Eichhorst, and suggest that the fatty degeneration of the heart, which unquestionably follows section of the vagi, cannot be entirely accounted for either by pyrexia or starvation, but is referable to the disturbance of the normal influence which these nerves exercise upon the nutrition of the heart. The speculations upon the precise manner in which this influence is exercised need not be reproduced here.

THE STIMULATION AND CONTROL OF THE CARDIAC RHYTHM.—Several important points bearing upon these subjects have recently been investigated by Ludwig (of Pontresina) and Luchsinger, and are described in an elaborate paper in Pflüger's *Archiv*, Band xxv., s. 211. The most important conclusions alone require to be recorded here. 1. The vagi remain active in their inhibitory cardiac function, when an indifferent salt-solution is made to circulate through the heart instead of blood. 2. The same result is seen when a bland oil is substituted for blood. Thus the *blood-stimulus* as the prime mover of the cardiac activity is disproved. 3. The element of truth, however, which is contained in that expression is demonstrated by several experiments made by the authors. The *pressure* exerted upon the endocardium by the circulating fluid, whether blood, serum, salt-solution, or oil, was found to influence the frequency of the cardiac contraction directly; and that whether the whole heart, or the heart deprived of the sinus, or the isolated apex (that is, destitute of any ganglia, as far as is known) was tested. These results appear to be novel, although numerous facts have pointed for many years to the importance of the intra-cardiac pressure as the prime mover of systole. Coming next to the influence of the vagus in controlling the cardiac action, Ludwig and Luchsinger discovered that the higher the intra-cardiac pressure, the more difficult it is to arrest the heart by the vagus; not only the frequency but the force of the systole being increased by the rise of pressure. The variations in frequency depend entirely upon lengthening or shortening of the diastole, systolic time remaining unaffected. It must be observed, in conclusion, that these experiments, which furnish results apparently of the greatest value in clinical medicine, were performed exclusively on cold-blooded animals, chiefly frogs.

Vaso-Dilator Nerves.—MM. Dastre and Morat believe that

they are able to demonstrate in the dog the existence of vaso-dilator functions in certain branches of the sympathetic, which arise from the cord, and pass through the cervical ganglia to the bucco-labial region, the tongue and submaxillary gland, the ear, and less certainly to the upper and lower extremities. (*Centlbt. f. d. Med. Wiss.*, No. 20, 1881.)

The Innervation of the Salivary Glands.—The remarkably exact knowledge which we possess of the innervation of the sub-maxillary gland has been considerably extended by Aschenbrandt of Cassel, who has investigated the nervous relations of the other glands of the same class; the character of the different secretions furnished by the parotid, sublingual, and submaxillary glands, respectively; and the reflex relations of these structures to certain other parts of the body, notably the conjunctiva. The experiments were made on carnivorous animals. The principal results of the investigations may be summarised as follows:—Fluids that irritate the conjunctiva, cause salivation. The secretion of the parotid is influenced by the glosso-pharyngeal nerve, whilst the facial trunk has no share in salivation. The sympathetic is not influenced by irritation of the conjunctiva. All the three salivary glands participate in reflex salivation. The impression on the conjunctiva is reflected through the nuclei of the nerves. The character of saliva furnished by the different glands, and obtained by the various methods of reflex irritation, respectively, may be found described in the original paper in Pflüger's *Archiv*, Band xxv. s. 101.

Trophic Nerves and Nerve Centres (?).—Jarisch of Vienna has made the interesting observation that the anterior cornua of the cord were variously diseased in several cases of skin disease, namely, herpes iris, long-standing psoriasis, and acute lupus erythematosus. (*Centlbt. f. d. Med. Wiss.* 1881, No. 27, S. 504.)

Renal Centres in the Cord.—The subject of the influence of the spinal cord upon the amount and composition of the urine was originally discussed by Claude Bernard, and was subsequently investigated by several physiologists, notably Eckhard. Working in the laboratory of Professor Goltz of Strassburg, and enjoying there the great advantages of assistance and experience in making destructive lesions of the spinal cord, Mr. Barney Sachs, of New York, has reached certain important, although chiefly negative conclusions. His investigations were carried on chiefly on dogs

and rabbits. It is evident that section of the cord might influence the renal secretion in either, or both, of two ways; namely, first, directly, and secondly, indirectly through the circulation. Mr. Sachs finds, first of all, that no direct nervous influence upon the activity of the kidneys can be attributed to the spinal cord. In whatever region it is cut, no distinct effect is produced upon the secretion of urine, at least to any degree. Neither does there seem to exist any definite relation between the blood-pressure and the amount of urine. Whatever centres there may be for the kidneys, they certainly do not exist in the cervical portion of the cord, between the third cervical and the second dorsal vertebræ, and the presence of fibres passing through the cord from a higher centre is quite unproved. (Pflüger's *Archiv*, Band xxv., S. 299.)

J. Mitchell Bruce, M.D.

Israel (Berlin), **Case of Gunshot-wound of the great Brachial Nerves, with consecutive Atrophy of the Arm.**—Dr. Israel (Virchow's *Archiv*, vol. lxxxv. 1) gives an account of the post-mortem examination of a man who, in March 1848, had received a shot in the region of the left shoulder. The bullet made its entrance at the lateral margin of the pectoralis major and came out in the lower third of the scapula. In the last six months of his life, the patient had exhibited symptoms of chronic brain-disease: apoplectiform fits, headache, paralysis of the left facial nerve, mental disturbance. In September 1880 he had repeated attacks of giddiness, tremor artuum, especially in the left arm, vomiting and singultus. He died in a fit of this kind, September 9th, 1880. On examining the patient four weeks before his death, Dr. Brieger found the following condition of the left upper extremity:—Considerable atrophy of the whole arm, but particularly of fore-arm and hand, thumb extended. Passive movements in the joints of left thumb impaired. Active mobility of shoulder, elbow and wrist perfectly free. Second and third phalanges in flexion, enormously atrophied. Tendons in the palm distinctly discernible on account of atrophy. Flexion and extension of hand free; abduction and adduction impossible. The difference of circumference of the arms varies from 2–3½ centimetres; difference of temperature from 0·6–3·3 centigrade in the different parts of the extremities, the greatest difference being in the palms of the hand.

Direct electric excitability of biceps, triceps, deltoid and extensors of the hand normal. Direct electric irritability of the left ulnar nerve, faradic as well as galvanic, entirely abolished. Tactile sensibility of left palm distinctly diminished; perception of tem-

perature slightly so. Perception of pain not impaired. Otherwise no alteration of sensibility to be found.

The autopsy revealed a tumour in the centre of the left hemisphere, chiefly occupying the insula and part of the third frontal convolution. Internal capsule and pyramidal strands intact, nucleus lentiformis partly affected. In the spinal cord the substance of the middle and lower part of the cervical region distinctly asymmetrical, the left side being considerably smaller than the right.

Muscles of left upper arm a good deal atrophied; those of fore-arm emaciated in the highest degree. There is a cicatrix of 3 cm. to 0·5 cm. on the lateral margin of the pectoralis major. From this cicatrix strings of connective tissue penetrate the muscles, crossing the great brachial nerves. The ulnar and median nerves form a regular loop, ending in the cicatrix; they unite by means of an intermediate piece of nervous tissue; each of them shows an intumescence of 2 cm. length, and 1·5 cm. in diameter. Both nerves have the diameter of a pencil. The radial nerve is but slightly entangled in the cicatrix. In striking contrast to this condition, the peripheral parts of the ulnar and median nerve are considerably atrophied, their diameter being that of a knitting-needle. Having passed through the fascia of the arm, they enter a direct union with the cutaneous median nerve, which appears thicker than usual. Microscopically, the central termination of both nerves shows real nervous tissue, whereas the peripherical parts consist chiefly of connective tissue.

It need hardly be mentioned that the alterations of the left arm were due solely to the injury, and not to the cerebral tumour. The integrity of sensibility, which was almost perfect, is explained by the communication of the injured nerves with the cutaneous median nerve. It is a remarkable fact, however, that, in spite of this detour, the sensory impressions in the ulnar region were localised in the same way as before the injury, whereas the centrifugal action of the motor nerves had not been restored.

Nothnagel on the influence of Cerebral Injuries on the Reflexes.—Prof. Nothnagel (*Zeitschrift f. Klin. Med.* iii. 2) has made some experiments on rabbits and pigeons in order to find out whether there are in warm-blooded animal centres of inhibition for the reflex movements analogous to those of the frog discovered by Setchenow. He irritated the hemispheres either by destruction of a part of the substance of the brain by means of a strong needle,

or else, by stimulating it with a faradic current, which was conducted to the brain by the points of two platinum wires. In one instance, half a Pravaz' syringe filled with a mixture of paraffine and grease was injected into the brain of a rabbit through a trephine hole. In this case the animal became unconscious, the action of the heart was considerably slackened, respiration ceased, and all reflexes, including even corneal reflex, were abolished for a few minutes. In all other cases no alteration of the reflexes was observed either during, or after, the stimulation or injury of the brain. The author, therefore, has arrived at results very different from those of Simonoff and Goltz, who found in dogs a considerable diminution of reflex action after mechanical and electric stimulation of the cortex.

Strümpell on Apoplectic Bulbar Paralysis. (*Neuropathologische Mittheilungen, Archiv für klin. Med.* xxviii. 1.)—A man, æt. 36, previously in good health, was suddenly taken ill with a violent fit of vertigo, followed by a sensation of excessive heat in the left side of the whole body. There was no loss of consciousness, no headache, no paralysis in the trunk or extremities, but a slight impairment of the movements of the facial muscles. The most prominent symptom, however, was the absolute impossibility of swallowing. During the first two or three hours after the attack the head was several times drawn to the right side. No other spasmodic symptoms were observed. The patient was bled at once, and had an ice-bag applied to the head; this latter was felt by the patient as warm on the left side, and cold on the right.

Three days after the attack, the author noticed the following condition of the man:—Temperature normal; pulse 56. Complete paralysis of the right facial nerves. Ptosis of the left eyelid, slight nystagmus.

Tongue deviates a little to the right. The patient feels some difficulty in the movements of the tongue. No impediment of speech. Soft palate, perfectly normal, uvula in the normal position. The patient is unable to swallow anything, either liquid or solid.

Slight strabismus internus of the right eye.

Analgesia of left side of face. Differences of temperature are well perceived on the left side of the face, but everything produces here a sensation of heat. The same phenomenon is observed in the left side of the trunk and both left extremities. No other abnormality to be found.

The patient was galvanised at once; anode in the nape of the

neck, kathode in the region of the larynx. The current is frequently interrupted, and voltaic alternatives applied.

After seven weeks of this treatment the patient began to regain the power of swallowing a little water, and after another three weeks he had quite recovered. Three years and a half have passed since then, and the man is still perfectly well. During his sojourn at the hospital, slight hæmorrhages of the skin of the forearm and in the conjunctivæ of the right eye were observed.

Dr. Strümpell points out that this remarkable case cannot be explained otherwise than by a lesion in the bulb and posterior part of the left side of the pons. There was slight paresis of the right hypoglossus, as shown by the difficulty of the movements of the tongue; irritation of the accessory nerve (the head being drawn to the right side); by the retardation of the pulse irritation of the vagus was indicated. These were the bulbar symptoms. The paralysis of the left facial nerve, paresis of oculo-motor nerve (ptosis) and abducens (strabismus) indicate that the pons was also influenced by the disease. All these symptoms, however, were but of a transitory character, while after a short time the only remaining symptom was the paralysis of the swallowing-movements.

As to the character of the lesion, the author thinks that it was most probably a hæmorrhage, there being evidently in the patient a tendency to hæmorrhagic diathesis, as shown by the spontaneous effusions of blood in the arm and eye.

Dr. Strümpell gives, in addition to the above described case, a short account of two other observations of apoplectiform bulbar paralysis.

The first case was a female, 56 years of age. Six years before the present illness she had had a sudden fall from a staircase, since which she had shown a certain weakness in her right leg. She had been suffering from occasional fits of giddiness and headaches; for the last six months she spoke somewhat less distinctly than usual. On the 14th of August, 1877, the patient was found in a state of absolute speechlessness; she indicated by signs that this had come on suddenly at 2 o'clock in the night, without the slightest loss of consciousness. Three days afterwards she began to have difficulty in swallowing. Four days later she fell down in a new attack of giddiness; there was, however, nothing remarkable to be observed after this attack. On examining the patient on the 25th of August Dr. Strümpell found complete paralysis of the tongue; paresis of the soft palate, great difficulty of swallowing. After a few days

the patient began to regain the power of pronouncing several letters more or less distinctly. The tongue became a little moveable. Pulse constantly retarded. The patient walks slowly and cautiously, the right leg being somewhat dragged.

In September 1880 the following condition was stated: Patient out of bed all day; walks with short steps, mostly supporting herself by the bedstead. Constant salivation. A certain degree of dementia is observed. No atrophy of lips. Tongue not atrophied, no fibrillary contractions. Swallowing impaired. On laryngoscopical examination, paresis of the vocal cords is found. Language all but incomprehensible, low, anarthria. Galvanic and faradic contractility of tongue perfectly normal.

In the second case, a man, 56 years of age, who had always been in good health, broke down in the road, having worked all day in cold rainy weather. When he was brought home, he soon felt better, but he observed that "his upper eyelids dropped down gradually." Two months later he was seen at the hospital. There was ptosis of both upper eyelids, paralysis of left rectus internus, slight weakness in both arms. Within the following month progressive muscular atrophy in both arms, gradually increasing impairment of speech and swallowing, paresis of the lips, weakness of the legs, were noticed. Two months after his reception at the hospital, the man died in a fit of violent dyspnœa, there being complete impossibility of expectoration.

At the autopsy, brain and spinal cord showed not the least macroscopical alteration. On microscopical examination Prof. E. Wagner (Leipzig) found several small hæmorrhages in the nucleus of the third nerve, and in the medulla oblongata numerous little cavities, partly corresponding to very small aneurisms.

Strümpell: Case of Brachiofacial Monoplegia with Disturbance of Speech.—Recovery.—A widow (æt. 27), who had never been ill, but had had a good deal of sorrow and anxiety within the last year, observed suddenly a slight difficulty in speaking. A few days later she began to feel a certain weakness in the fingers of the right hand, which gradually increased, so that about three weeks afterwards the right arm was completely paralysed. There was also a slight paresis in the right side of the face.

A month after the commencement of her illness the patient was received at the hospital. There was paresis of the inferior part of the right facial nerve; complete paralysis of the right arm; sensibility normal; no skin reflexes, exaggeration of tendon-reflexes

in right arm. Electric irritability normal. Peculiar disturbance of speech. The patient avoids speaking as much as possible; she talks slowly and carefully. She puts in wrong letters, or leaves out a letter, or mistakes one letter for another. In pronouncing repeatedly the same word, she learns to avoid the mistakes. She is easily fatigued by talking, and then makes more mistakes than in the beginning. Occasionally fits of tonic spasm on the right side are observed. They always begin in the right leg by a sensation of tingling, which ascends to the right arm, and is followed by tonic spasm of the inner three fingers of the hand, index and thumb remaining extended. When this is over, a similar sensation takes place in the right cheek, followed by a tonic contraction of the inferior muscles of the right side of face. Compression of the blood-vessels and nerves of right arm have no influence on these spasms. Clonic contractions never took place. By degrees the patient got better, and four months after her reception at the hospital (May 1879) she was dismissed perfectly cured, and remained so to this day. The only anomaly to be observed now is a certain cautiousness in speaking and a very slight want of innervation in the right facial nerve. The disturbance of speech is characterised by the author as "literal ataxy;" just as in locomotor ataxy the movements are disturbed by involuntary movements interfering with those that are intended by the patient; in this case letters which were not wanted for the formation of words were put in irregularly, and prevented the formation of normal words. The tonic spasms were symptomatically exactly like those of tetanus; they differed essentially from the usual epileptiform clonic spasms which are commonly noticed in cortical lesions.

Dr. Strümpell considers the case as one of curable encephalitis in the motor region, more particularly in the region of the middle and lower third of the ascending frontal and parietal convolutions, which has been shown by Ferrier to be the centre of the movements in question.

R. H. Pierson, M.D.

Straus on Tabetic Ecchymoses.—Straus (*Archives de Neurologie*, No. 4, 1881) describes another form of cutaneous eruption succeeding the lightning pains of ataxy, in addition to the papular, pustular, herpetic, &c., eruptions described by Charcot and Vulpian. These consist in veritable ecchymoses, which appear in a certain number of ataxics after the cessation of a violent accession of pains, sometimes not showing themselves for several hours. The appear-

ance and course which they pursue correspond entirely with the extravasations resulting from injury, and were at first supposed by the author to be really due to mechanical injury, but which he found on careful and repeated examination not to be the case. The spontaneous nature of the ecchymoses was clearly proved. The patches are irregular in shape and size, and also variable in number. The intensity of the discolouration is generally proportional to the duration and violence of the pain. They almost always occupy the member, or part of it, which has been the chief seat of pain, and may be unilateral or bilateral accordingly. Generally the ecchymoses occur at a higher level than the actual seat of pain; in the leg, if the ankle has been specially attacked, and in the upper arm, if the elbow has been the seat of the pain.

The distribution of the patches does not correspond with the course of the cutaneous nerves like the tabetic eruptions described by Charcot. Sometimes, but exceptionally, the ecchymoses are confined to one limb, and it may happen that they appear not in the limb which has been the seat of pain, but in the opposite. No particular period in the course of the disease can be fixed at which they occur. Nor can they be said to be very common.

As to their mode of causation, mechanical injury being excluded, two hypotheses may be entertained. According to the one most in harmony with established data, the ecchymoses may be looked upon as the result of local vascular dilatation, either active vaso-dilatation or passive vaso-paralysis, caused reflexly by irritation of the posterior radicular zones acting on the anterior roots. The other hypothesis is that the ecchymoses are due to direct irritation of vaso-dilator fibres contained in the posterior roots. Most physiologists hold that the vaso-motor nerves pass out by the anterior roots, but the experiments of Brown-Séquard seem to indicate that the posterior roots also contain vaso-motor fibres. More recently, Stricker has published experiments which he holds demonstrate the existence of vaso-dilator fibres in the posterior roots of the sciatic in the dog. This view has been contested by Cossy and Vulpian, but again affirmed by Stricker. If Stricker's view is correct, then the ecchymoses would find an explanation in the direct irritation of vaso-dilator nerves of the posterior roots by the active process on which the lightning pains depend.

Debove on Tabetic Arthropathies.—Debove (*Archives de Neurologie*, No. 5, 1881), in remarking on a case of tabetic arthropathy,

calls attention to the multiplicity and symmetry which these affections exhibit in the individuals attacked. In his case the left elbow and both shoulders were affected—the arthropathy of the right shoulder showing itself first after a mechanical injury.

The liquid removed by puncture corresponded in all its characters with synovial fluid. Charcot and other observers have noted the suddenness with which the swelling of the joint and its neighbourhood occurs, and this, according to Debove, is the result of the escape and infiltration of the cellular tissue by the synovial fluid, which causes swelling and enlargement of the superficial veins by pressure on the deeper ones. The extent of the swelling is conditioned by the arrangement of the cellular tissue and fasciæ.

As to the mode in which the articular fluid escapes, it is not the result of arthritis and slow distension, but more probably the rupture and inflammation are simultaneous.

Owing to this rupture and absence of distension in the joint, is due, in part at least, the freedom from pain which is so characteristic of this form of arthropathy. As to how the rupture occurs, it does not seem due to direct rupture of the capsule, as affections of the ligamentous tissues are not characteristic of ataxy. The author believes that it can be most satisfactorily accounted for by the same degeneration of the osseous tissue which leads to the spontaneous fractures of tabetics. This, occurring at the attachments of the capsular ligaments, would lead to the escape of the synovial fluid and the resulting tumefaction. An examination of the joints in tabetic arthropathy also supports this view, as the lesions of the articular surfaces are often unequal. In true arthritis the whole articular surface is involved in the inflammatory process.

D. Ferrier.

// BRAIN.

JANUARY, 1882.

Original Articles.

ON THE POSITION OF THE MOTOR CENTRES IN THE BRAIN IN REGARD TO THE NUTRITIVE AND SOCIAL FUNCTIONS.

BY T. LAUDER BRUNTON, M.D., F.R.S.

In one pregnant sentence the German philosopher Goethe sums up the essence of human action. "Warum treibt das Volk so und schreit? Es will sich ernähren, Kinder zeugen, und sie ernähren wie es vermag." (Why are people so busy, and what are they roaring about? They want to feed themselves, to beget children, and feed them as best they can.) In these three reasons for human activity we find that one is personal and that two are social. The first of these is the personal one, the duty of the individual to himself. The second and third are the social ones, the duties of the individual to society. The clear insight of the philosopher led him rightly to put the duty of the individual to himself first. Under certain conditions we find that the social may overpower the personal instincts, even in the lower animals, and lead the mother to sacrifice her own life in trying to savê her progeny. In certain exceptional instances we find the social instinct extending beyond the circle of immediate blood-relations, and inducing a man, as in the well-known story of Damon and Pythias, to sacrifice his life for that of his friends, or even inducing him, like Sir Philip Sydney, to refuse the water which

was offered to quench his own burning thirst, and give it to an utter stranger simply because he believed the other to need it more. Such instances as these do occur; but the care with which they are recorded, and the admiration with which they are regarded, clearly show how exceptional is their occurrence. The instinct of self-preservation is one which underlies, and, except in the very rarest cases, is more prominent than the social instincts. It may sometimes be brought out only in the utmost need; but when the survivors of a shipwreck are struggling for the last particle of food on board a raft, or for the last drop of water in the midst of the Sahara, or for a breath of air in the Black Hole of Calcutta, then the instinct of the individual to preserve himself, whatever may be the cost to others, makes itself manifest. The instinct of self-preservation, indeed, must be the fundamental one, for otherwise the evolution of the races of animals now living would have been impossible. Animals not having this instinct of self-preservation would necessarily have perished in the struggle for existence, and those endowed with it would have succeeded. The fundamental acts of self-preservation are the respiration of air and the acquisition of food. Respiration is a simple act, and, as the air is everywhere around us, very simple rhythmical movements are sufficient to carry it on. These movements, too, require no further development during life, and the respiration of the newly-born child is as perfect as that of the full-grown man, as far, at least, as the aeration of the blood is concerned. No doubt the adult learns to modify his respiratory acts for such purposes as singing and speaking, and the nerve-centres by which these modifications are affected are capable of gradual development by careful training. They are no doubt to be sought in the cerebrum, but the nervous centre through which the mere aeration of the blood is effected co-ordinates very simple movements only, requires little or no further development, and is situated in the medulla oblongata and upper part of the spinal cord. The centres by which the primary acts of nutrition, deglutition, digestion, &c., are effected, are also situated in the spinal cord and in the sympathetic ganglia, for an anencephalous infant will suck, and a pigeon deprived of its brain will swallow the grains of corn

placed in its beak, but they are unable to seek their food. The centres by which the more complicated acts required in order to obtain food are, no doubt, situated in the brain. Hence the anencephalous infant, or the animal deprived of its brain, are unable to seek food for themselves, although they may swallow it when it is placed within their mouths. Modifications of respiration in producing voice, although exceedingly important for men and animals in their social relations, are comparatively unimportant for the nutrition of the individual, whereas the movements for seeking and obtaining food are essential to its existence. We should therefore expect that the motor centres in the brain would be arranged chiefly with reference to the acquisition of food, and that the centres would be modified in different animals according to the manner in which they obtained their food. That this is really the case appears, I think, from an examination of the functions of the motor centres of the brain as ascertained by Ferrier :

1. The eyes open widely, the pupils dilate, and head and eyes turn towards opposite side.

2. The opposite arm and hand extend forward as if to reach or touch something in front.

3. The opposite arm is retracted and adducted, the palm of the hand being directed backwards.

4. The fore-arm is supinated and flexed, so as to raise the hand to the mouth.

5. The angle of the mouth is retracted and elevated.

6. The ala of the nose and the upper lip are elevated, and the lower lip is depressed, so as to expose the canine teeth on the opposite side.

7, 8. The mouth is opened and the tongue protruded and retracted. The mouth is alternately opened and closed, and the tongue moves. The movements here are distinctly carried out bilaterally.

9. The opposite angle of the mouth is retracted, the platysma myoides is thrown into action, and when this is powerful the head is drawn slightly to the side.

(*a*), (*b*), (*c*), (*d*). Individual and combined movements of the fingers and wrist, ending in clenching of the fist. Centres for the extensors and flexors of the individual digits could not be

differentiated, but the prehensile movements of the opposite hand are evidently centralised here.

10. Advance of the opposite hind limbs, as in walking.

11. Complex movements of the thigh, leg, and foot, with adducted movements of the trunk.

12. Movements of the tail, associated with some of the last movements.

If we look at the accompanying diagram, copied from Ferrier's work, with an alteration in the numbers by which he designates the different centres, we see that in the monkey the centres appear to lie around the fissure of Rolando in a definite order, corresponding to that which would be necessary for the animal to obtain food. Taking the centres in the order in which we have numbered them, we find that, when they are successively irritated, the sequence of movements is as follows:—that the eyes open widely, the pupils dilate, and the head turns as if to look for food. Next the hand and arm are extended as if to take it. The arm and hand are next retracted and adducted. Next, the hand is supinated and the fore-arm flexed, so as to bring the hand towards the mouth, as if to convey food into it. Next, the angle of the mouth is retracted and elevated, the canine teeth are exposed, the mouth is opened, the tongue retracted and protruded, and the mouth alternately opened and closed with movements of the tongue as during mastication. There is next retraction of the opposite angle of the mouth, with powerful contraction of the platysma myoides. I do not know whether this has any connection with the emptying of the cheek pouches in the monkey, but it is certainly an accessory act in mastication and deglutition. It is not observable in man under ordinary circumstances, but it at once becomes evident when deglutition is rendered difficult by sore throat and tonsillitis. A similar action is also noticed in man when powerful movements of the jaws are required, as in cracking a hard nut.

The next movements (*a*), (*b*), (*c*), (*d*), are complicated movemements of the hand, which have not been more particularly analysed. Complicated movements of the hand are certainly required in frugivorous animals such as monkeys, in order to separate the edible parts of their food, as, for example, to pick out

FIG 1.

1. The eyes open widely, the pupils dilate, and head and eyes turn towards opposite side.

2. Extension forward of the opposite arm and hand, as if to reach something in front.

3. Movements of tail (and trunk).

4. Retraction with adduction of opposite arm.

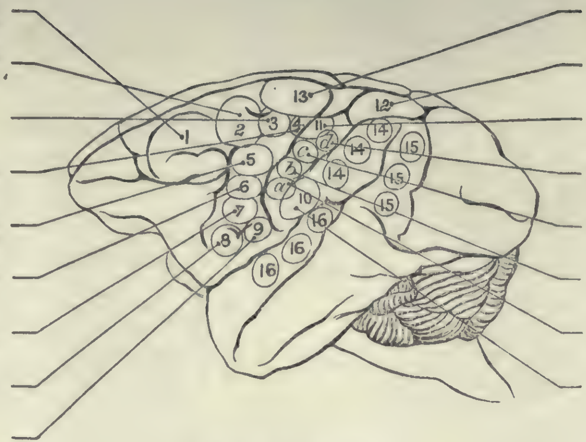
5. Supination and flexion of the fore-arm, by which the arm is raised towards the mouth.

6. Action of zygomatics, by which the angle of mouth is retracted and elevated.

7. Elevation of ala of nose and upper lip.

8. Opening of mouth with protrusion of tongue.

9. Retraction of tongue.



13. Complex movements of thigh, leg and foot.

12. Advance of the opposite hind limb.

11. Retraction and adduction of opposite arm.

a, *b*, *c*, *d*. Prehensile movements.

10. Retraction of opposite angle of mouth.

BRAIN OF MONKEY.

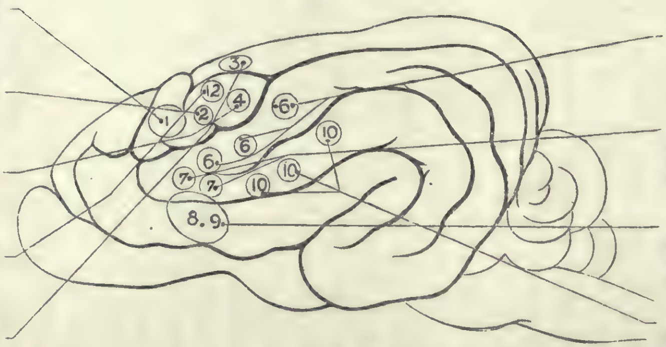
the kernel from a nut which they have just cracked. The centres are absent in the dog, where no such movements are wanted.

The next movement is that of retraction and adduction, and is the same as that which has already been noticed as being employed in gathering food. I have numbered the centre for this movement 11, and it is the same as that which I have numbered 4. Ferrier has reckoned these two centres as one. I have, however, taken the liberty of dividing his single centre into two, inasmuch as it extends over the fissures of Rolando, and as it appears to me that this movement, although of the same character, is employed for a double purpose. It seems to me that while the movement of retraction and adduction started by centre No. 4 is for the purpose of gathering food, the same movement is related, when started by centre 11, to the casting away of the refuse portions of food. I have not had an opportunity of observing the habits of monkeys since I began to write this paper, but, so far as my recollection serves me, the movement of the monkey in throwing away refuse is somewhat peculiar, nut-shells being cast by these animals, not to the right side, as they probably would be by us, by an outward movement of the hand, but down to the left by a quick movement of retraction and adduction. Next, the leg is advanced as in walking, (10) and then come complex movements of the thigh, leg, foot, and trunk, with movements of the tail in monkeys. These we may regard as movements of balancing, or possibly of climbing, in order to reach a new supply of food after the first has been consumed.

We have now finished the round of movements necessary for the procuring and masticating of food, and the cycle of movement again commences, with No. 1. On comparing the arrangement of centres in the brain of the dog or jackal with those in the monkey, we are struck by a curious difference. While in the monkey the centres are arranged in the order just mentioned around the fissure of Rolando in unbroken sequence, we observe that in the dog and jackal the centres which are arranged round the crucial sulcus, corresponding to the fissure of Rolando, are few in number, and that the others are situated lower down, on a different convolution. The

four centres surrounding the crucial sulcus in the dog, in Fig. 2, are numbered, in Ferrier's experiments, 12, 5, 4, and 1 respectively, corresponding to the centres numbered, in Fig. 1, 1, 2, 4, and 12 respectively. These centres are those for the direction of the eyes, movement forward of the fore-leg, its retraction and adduction, and advance of the opposite hind limb. These are the movements required for seeing food, and for simple running straight forward in active pursuit of the fleeing animal which is to supply it. The use of the lateral movements of the tail in pursuit is seen in the grey-hound, where the tail aids the animal in guiding its rapid course or in turning; but as it also is not so essential as the movements of the limbs, nor in such constant action, its centre, No. 3, Fig. 2, is placed somewhat aside from the principal gyrus. We see here that the arrangement of the centres in the dog and jackal differs from that in the monkey very much in the way we would expect, for while the monkey, living in trees yielding abundance of fruit, has only to see where the food is hanging and by one or two efforts to reach it, animals of the dog tribe have usually, after seeing their prey, to run for a considerable distance before they can hunt it down, and bring into action the muscles of the mouth and jaws by which they seize and devour it. The centres for sight and for running are, then, arranged in simple order round the crucial sulcus, while those for the movements of the jaws are placed in an adjacent convolution (Fig. 2). In his 'Expression of the Emotions,' Mr. Darwin has noted the action of the muscles of expression in protecting the eye from injury or strain, and one of the points that strikes us most forcibly in the arrangement of the motor centres in the dog is the action of the zygomatics and orbiculares in causing closure of the eyes, this closure being very closely associated with movements of the mouth. I do not know whether wolves or jackals shut their eyes at the moment they fix their fangs in the flesh of their prey, but such a movement as this would seem to be well adapted for the protection of their eyes, either from injury by the animal they attack, or from the effects of the increased tension caused by the muscular efforts in the act of attacking. On looking at the brain of the cat we are struck

Fig. 2.



Brain of Dog. (The numbers on the centres in the diagram correspond to those in Fig. 1.)

by the absence of the centre No. 12 in Ferrier's work, corresponding to No. 1 in the diagram in this paper, the centre which causes movement of the eyes. At first sight this seems very extraordinary, because the formation of the eyes of the cat is so peculiar that one is accustomed to think that it hunts its prey very much by sight. The want of this centre seems to me to render it probable that the sense of hearing and the tactile sense furnished by the cat's whiskers are considerably more important in directing the animal towards its food than even the sense of sight. We see, also, that the centre 4, for the retraction and adduction of the fore-arm, is much more developed than in the jackal or dog, and this we should conclude from the use which the cat makes of the fore-arm in striking its prey. On looking at the centres in rabbits, guinea-pigs, and rats, we see that they are arranged in the order 2, 4, 12, in this diagram, and 5, 4, 1 in Ferrier, that is to say, extension and retraction of the fore-leg, and forward movement of hind-leg. There is also great extension of the centres for the movements of the mouth, just as one would expect in rodents.

From this arrangement in these different animals it appears, I think, that the motor centres are arranged round the crucial sulcus or fissure of Rolando in such an order as, first of all, to subserve the wants of the animal in obtaining food, the motor centres which are first called into requisition in going after the food being nearest to the fissure, and those which are only required after the first centres have been in action for some time being arranged in an adjoining convolution. The social instincts, and especially the sexual, which is the primary social instinct, are subserved more especially by those modifications of respiration which give rise to voice. We find animals of the cat tribe purring or growling over their food, yet the voice is not of primary importance in obtaining food. Its chief use is that of enabling the animals to communicate with each other. Such communication is no doubt primarily of sexual importance, but it is also useful in enabling animals of the dog tribe, such as wolves, to obtain food by summoning one another to collect together for the purpose of hunting in packs. Although there are some very marked exceptions

amongst animals, it is, generally, only after the wants of the individual have been satisfied by food that the sexual instinct comes into play, and we should therefore expect that the motor centres connected directly or indirectly with it, such as the centres for voice, would be further removed from the crucial sulcus, or fissure of Rolando, than the motor centres required for the obtaining of food. This we find to be actually the case, but I am not at present in possession of sufficient data to enable me to extend my paper to this branch of the subject.

Even should the conclusions at which I have arrived, and which I have here stated, be erroneous, the hypothesis that the motor centres are arranged in the way I have described will enable those who have experienced difficulty in remembering their position to do so with ease, as I have had occasion to observe in the case of students.

For convenience of reference I give here in tabular form the corresponding centres as numbered in Ferrier's work on the Functions of the Brain, and in Figs. 1 and 2 in this paper.

Ferrier.	Figs. 1 and 2.
1	12
2	13
3	3
4	4
5	2
6	5
7	6
8	7
9	8
10	9
11	10
12	1

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

BY BEVAN LEWIS, L.R.C.P. LOND.

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(Continued from p. 360.)

Examination of Internal Structure of the Brain.

1. INTRODUCE a large section knife into the longitudinal fissure, and cut outwards across each hemisphere, about ¾ inch above the corpus callosum, exposing the *centrum ovale minus.* Repeat the same procedure on a level with the corpus callosum, exposing the greater centrum ovale, and exhibiting the continuation of the transverse strands of this great commissural tract with the medulla of the hemisphere. The student will now proceed to note the relative and absolute amount of grey and white matter, and inquire into the consistence, colour, and other physical qualities of these parts in the manner already detailed.

2. By an incision carried from before backwards across the fibres of the corpus callosum, a short distance on each side of the median line, the lateral ventricles are opened. To expose the structures more fully, divide the corpus callosum anteriorly and whilst gently reflecting it cut backwards, with a scissors through the vertical septum (septum lucidum), which is attached to its under surface, and which, descending, divides the lateral ventricles into two cavities. After reflecting the corpus callosum and dividing across behind, note the condition of the following structures seriatim—

a. The caudate nuclei or intra-ventricular portions of the corpora striata.

b. Septum lucidum, enclosing betwixt its walls the fifth ventricle.

c. Exposed anterior tubercle of the thalamus opticus.

d. Stria cornea, or exposed part of the tænia semicircularis coursing between the thalamus and corpus striatum.

e. Fringed margins of the velum interpositum or choroid plexus of lateral ventricles.

f. Fornix, and behind its descending pillars, the foramen of Monro.

g. General condition of lining membrane or ependyma.

3. Divide the fornix anteriorly close to its descending pillars, and after examining the subjacent velum interpositum, reflect both structures backwards, exposing the thalami optici, separated by the intervening third ventricle, and note the condition of the following parts:—

a. Grey matter forming the boundaries of the central cavities.

b. Anterior, middle, and posterior commissures.

c. Pineal gland and its ganglionic extensions, or peduncles.

d. Corpora quadrigemina (nates and testes).

e. Follow out the fornix posteriorly into the descending cornua of the ventricles.

4. On separating the occipital lobes from the cerebellum the curved bistoury may, by a little careful dissection, be made to cut through both *crura cerebri* obliquely upwards, to meet *above the corpora quadrigemina* at an obtuse angle in the third ventricle in front of the posterior commissure.

By so doing we separate from the cerebrum the cerebellum, pons, and medulla, together with the *corpora quadrigemina* and *pineal gland.* This method is certainly preferable to dividing the crus at the level of the superior cerebellar peduncle, which is so often done at the sacrifice of the normal relationships of most important regions.

5. Separate the cerebral hemispheres by median section through the slight remaining connections, and proceed thus:

a. Slice one of the hemispheres in a direction parallel to the sections already made when exposing the centrum ovale. Let numerous sections in this direc-

tion be made at different planes from above downwards through the whole hemisphere, so as to expose the structure of the thalamus and corpus striatum down to the base.

b. Slice the remaining hemisphere in a direction at right angles to its long axis, or, in other words, from above downwards, beginning on sections near the olfactory bulb, and proceeding as far back as the occipital lobe.

The sections (*a* and *b*) will familiarise the student with the *relationships and coarse structure* of the basal ganglia, points of considerable moment. Examine, therefore, by aid of these sections the medullary tracts forming the *internal and external capsules*, the *lenticular and caudate nucleus* of the corpus striatum, the *thalamus opticus* and its various regions and environment. All these parts may be studied in succession, referring to good illustrations, such as appear in the last edition of Quain's 'Anatomy.' The intimate structure and relationships of these regions will be dealt with further on.

6. Remove the cerebellum from its attachments by dividing the three peduncles, and proceed to examine successively

a. Superficial aspect of membranes covering pons and medulla.

b. Condition of blood-vessels and superficial origin of the cranial nerves.

c. Condition of valve of Vieussens, aqueduct and fourth ventricle.

d. By transverse sections expose structure of nates and testes, of the pons, its anterior, or motor and posterior or tegmental tract, of the medulla oblongata, noting the relative dimensions and appearance of the olivary bodies, the pyramidal and restiform tracts.

7. Lastly, after examining the lobules and membranes of the cerebellum, divide it into an upper and lower half by an incision carried through both hemispheres from behind forwards, exposing the central medulla.

Vertical section, i.e. at right angles to the lamina of the cerebellum, will display its foliated arrangement, and betwixt vermiform process and the middle of each hemisphere we pass through the plicated corpus dentatum.

Minute Examination of the Brain.

Prior to considering in detail the methods adopted for the minute examination of the brain, the student should be fully impressed with the conditions which render many of the processes applicable for other tissues quite inadmissible here. The consistence of cerebral tissue is such that cutting fine sections by the hand, either with an ordinary razor or the Valentin blades, cannot be adopted with any hope of success, since the finest sections thus obtainable are useless for microscopic examination, which requires sections of extreme tenuity. These extremely fine sections can only be obtained by aid of the screw microtome from portions of hardened or frozen brain. In order to modify the consistence of the tissue so as to adapt it for this purpose, various methods of hardening by chromic acid, chrome salts, alcohol, picric acid, and osmic acid have been devised. The results which have accrued from these methods have been most valuable, yet they are uniformly open to the serious objection that this altered consistence induced by the reagent is obtained at the expense of modified structure and altered relationships. Amongst the most serious drawbacks to the use of corrugating reagents in the examination of the brain are the following :—

A considerable expenditure of time (from three to eight weeks being required for satisfactory hardening).

The process itself is extremely tedious, and often unsatisfactory in its results, as evidenced by experienced histologists.

It requires a considerable amount of practised manipulation, and exposes the tyro to numerous disappointments and failures.

The shrinking of tissue is a most objectionable feature in the cortex, often amounting to one-half the original bulk.

The normal wealth of structure is greatly modified ; in this respect it is infinitely surpassed by fresh methods.

The absolute and relative depth of the various layers of the cortex is subjected to most objectionable modifications.

It affects the cortex of different animals to a different extent, thus interfering with comparative investigations. Many pathological and normal appearances are wholly obliterated by the "hardening processes."

Of all tissues, that of the brain, from its extreme delicacy and susceptibility to rapid post-mortem change, is the tissue which, above all others, demands a rapid, ready and fresh method of preparation, the employment of indifferent media, and the restriction of all corrugating reagents. No method as yet adopted can surpass for elegance, expedition and certainty the freezing method; and, above all, it is *the* method which should be chiefly trusted to in all exact anatomical and pathological research. Yet, whilst we claim for the fresh freezing methods such self-evident advantages over the older processes, the long series of objections to the latter tabulated above must not induce us to blind our eyes to the fact that it also possesses its own advantages and cannot be dispensed with. Most of our classical descriptions of cerebral structure refer to brain which has been subjected to these methods of preparation; and few indeed, far too few, are the delineations of the minute structures of the cortex and medulla in the perfectly fresh brain. Hence, as a question of simple comparison between the results of different observers and our own, the hardening methods must still be adopted. There are numerous other very apparent reasons why we cannot afford to dispense with the older processes, and the student is therefore recommended, whilst guarding himself from its fallacies, checking or confirming its results by the fresh methods, to make himself equally familiar with the practical details of both methods, and to learn to discriminate the special worth to which each process is more particularly suited.

Two distinct methods of Freezing.—Just as we find amongst the list of reagents used for hardening brain some, and especially osmic acid, far less open to the objectionable qualities possessed by alcohol and chromic acid, inducing in fact very slight alteration in the relationships and bulk of the tissue, so do we find the different methods of freezing possess their own distinct and relative merit. This fact, which it is all important to recognise when dealing with structures like the

brain, has been wholly misunderstood or overlooked by all authorities who have written upon the subject, and we constantly find histologists grouping the freezing by ice and salt with the ether-freezing method, as though both were equally suitable for all tissues alike. Now the fact is that the ordinary *ice and salt* freezing-microtome is entirely useless for brain-structures, except after a modification of the latter by a process of hardening, and hence no longer a *fresh* method. Upon the other hand, the ether-freezing microtome is applicable to all tissues having a consistence not above that of the liver, but is pre-eminently adapted for nervous tissue. I find it necessary to insist upon this point, since it seems generally understood that the ice-and-salt mixture is suitable for freezing brain. Prior to the introduction of freezing by ether, practised histologists constantly complained of the impossibility of obtaining fine sections of brain by freezing, the objections being that hard spiculæ formed within its structure, and tore it up on cutting through it. Very lately this fact has been again asserted by a skilled manipulator,[1] who has even introduced a method of combined hardening and freezing to overcome this very unsatisfactory action. Hitherto, therefore, it may be asserted that the use of the ice and salt microtome has failed for the purposes of the cerebral histologist; and although by further development it may be rendered subservient to his purposes, for the present the freezing of brain substance must be uniformly pursued upon the ether-freezing microtome. The methods available for the examination of the minute structure of the brain are as follows :—

Hardening processes for sections to be cut on the imbedding microtome.

Hardening processes for sections to be cut on the freezing microtome.

Fresh process by means of the ether-freezing microtome.

Fresh process by a modified teazing and staining.

Fresh process by ordinary teazing with dissociating reagents.

We will preface our account of these methods by a brief outline of the more useful microtomes used.

[1] Vide Hamilton in the 'Jour. Anatomy and Phys.' vol. xii. page 259.

Microtomes for Imbedding.

Amongst the various instruments devised for cutting sections of hardened tissues are the microtomes of Henson, Rivet-Leiser, Brandt, Roy, His, Ranvier, Stirling and Rutherford. The instrument more generally used in this country is that devised by Stirling, and its modification by Rutherford, either of which instruments will prove satisfactory to the student; whilst that of Ranvier may be employed where it is desirable to grasp the microtome in the hand, or immerse it during section-cutting in water or spirit. The principle adopted in Stirling's instrument has been very generally followed. In the three last named, the body of the instrument consists of a metal tube or hollow cylinder, fixed to a table by some simple arrangement, or held in the hand in an upright or vertical position. A fine micrometer-screw works into the lower end, driving through the hollow of the cylinder a closely fitted piston-plug, which in its turn propels the mass in which the tissue to be cut is imbedded. The essential portion of this form of microtome, therefore, is the body, or cylinder, and the powerful micrometer-screw. The body, or cylinder, includes the following divisions:—the hollow, or 'Well' of the cylinder; its closely fitted plug, or 'Piston;' the upper smooth and levelled extremity, or 'Section-plate;' the lower extremity forming a "female screw" for the reception of the micrometer-screw. It is far preferable that the cylinder should be clamped by projecting arms and screw to a firm support as a table. In choosing a microtome for cutting sections of an imbedded tissue the student must be guided by the following considerations. The instrument should be strong but compact, and not weighty or cumbersome; it should possess appliances for fixing it firmly and immovably to a supporting ledge or table. Its well should be at least one inch in diameter, and the oval section is to be preferred to the circular well, since this form of well wholly prevents rotation of the imbedded mass. The section-plate should be absolutely level, perfectly smooth, polished, and show no irregularities of surface or indentations around the margin of the well, which otherwise would ruin the edge of the blade. The micrometer-

screw should be of powerful leverage, work evenly, easily, and without the slightest "loss of time," and have a pitch of at least fifty threads to the inch. Allowing his judgment to be guided by the above rules, the student cannot err greatly in his selection of a microtome for ordinary work and the smaller class of sections. A few words on the different varieties of instruments used may prove of service to the student.

Stirling's Microtome is a compact, strong, and admirably finished instrument, embracing all the qualities essential for the section-cutting of hardened preparations. For small sections no better adapted form has yet been devised, and for his earliest attempts in cutting hardened brain the student is recommended to secure this form of microtome. This instrument is made to be clamped to a table, so that both hands are free for section-cutting.

Ranvier's Microtome.—This is a smaller but very useful instrument. It has, however, to be held in the hand, a defect which is in part counterbalanced by the ease with which it can be immersed in spirit, and sections cut whilst so situated. It is well adapted for sections of spinal cord and the large nerve-trunks.

Rutherford's Microtome.—This, which is a modified form of Stirling's microtome, is a most valuable instrument, as it is well adapted for section-cutting of imbedded or of frozen tissues. It will be described fully in the section on freezing-microtomes. It has the disadvantage of being somewhat cumbersome as compared with the smaller "Stirling," whilst at the same time as a freezing instrument for nervous tissues it is excelled by the ether-freezing microtomes. The latter remark does not apply to other animal tissues.

Roy's Microtome[1] consists of an inner vertical brass plate covered with a layer of cork, and sliding by vertical movement within an outer brass-framework. A glass rod of horse-shoe form is fixed horizontally by its extremities into the brass frame, upon which the blade glides during section-cutting. The imbedding mixture is first cast in a zinc mould, the tissue being placed in it, and when hardened, the mass is fixed upon the cork plate. The vertical movement of the brass-

[1] Described and figured in the 'Journal of Physiology,' Vol. 2, No. 1.

slide is obtained by propulsion from a fine-threaded screw fixed below. A simple tubulature, adapted to the instrument, allows spirit or water to be blown upon the mass when required. It has been stated that tissues may be cut fresh with this microtome after freezing by the direct application of the ether spray. The student must be cautioned against any such attempt; for all tissues alike, the direct application of ether is to be deprecated, and for brain and spinal cord and all nervous structures such a method is wholly inadmissible. Roy's microtome has the disadvantage of not being readily fixed to a table during manipulation.[1]

Schiefferdecker's Microtome is described here as applicable to hardened preparations, although it is essentially constituted to dispense with the method of imbedding. The object to be cut is fixed within a hollow brass cylinder by means of a clamp upon its surface worked by two screws. In lieu of a propelling screw below, as in other microtomes, a circular plate forming the section plate can be elevated or lowered by the revolution of an outer concentric plate which turns upon a screw. The student will find this microtome figured and described in the 'Quarterly Journal of Microscopical Science' for January, 1877.

Microtome for slicing through whole hemisphere of Human Brain.—The instrument which I have made for this purpose at the West Riding Asylum consists of a heavy brass cylinder, three inches deep and eight inches in diameter, closed in below, where, however, its cavity communicates centrally with that of a small secondary cylinder—the screw-socket. In this socket, which is four inches deep, works a powerful and finely threaded screw, having a milled head three inches in diameter. A flat, circular brass plate, accurately fitted to the interior of the upper cylinder or well, is raised or lowered by means of the screw, the movement being communicated to the imbedding mass, which rests upon it above. The section plate is contuted by the projecting rim of the cylinder above, and is mathematically level and smoothly polished. The microtome rests upon the iron collar of a powerful tripod stand. A zinc

[1] The instrument is made by Mr. Gardner, South Bridge, Edinburgh.

tray, two and a quarter inches deep and twenty-six by seventeen inches, can be adapted to the microtome-cylinder, so that sections may be cut under water with the greatest ease.

Microtomes for Freezing.

These, as before stated, are either ether, or ice-and-salt freezing instruments.

The Ether-Freezing Microtome (Bevan Lewis).—This microtome, which I believe is pre-eminently suited for the purpose of the cerebral histologist, was described in the pages of the 'Journal of Anatomy and Physiology' for April 1877, and as modified and improved twelve months later, in the October number of 'BRAIN,' to which I must refer my reader for a detailed account of the instrument and the method of freezing. The section plate is either made of metal, or consists of plate-glass drilled with a central aperture through which the freezing chamber glides. I would strongly recommend the instrument which has the plate-glass top, as it forms a great safeguard against injuries to which the section-blade is subject from scratches and indentations on the metal surface. A further improvement has been introduced by the maker, in the form of a lateral support for the ether-spray apparatus, communicating with a bellows worked by the foot. In the latter form the spray adjustment is easy, and the hands are both free for section-cutting and manipulation, and thus a most substantial and valuable improvement has been obtained with but a very trifling addition to the cost of the microtome.[1]

Rutherford's Freezing Microtome.[2]—This instrument consists of a brass section-plate, with a central aperture leading into the interior of a vertical tube, in which a plug is fitted to move upwards or downwards by means of a fine screw. The cylinder is surrounded on all sides by a metal box covered with gutta-percha, which holds the freezing mixture of ice and

[1] These improved microtomes may be obtained of Mr. Gardner, 45, South Bridge, Edinburgh, who, at a minimum of charge, has fully perfected the workmanship.

[2] "A New Freezing Microtome." 'Monthly Micros. Journal,' vol. x. page 185. 'Outlines of Practical Histology.' By William Rutherford, M.D., 2nd edit. page 164.

salt. An exit tube allows of the escape of water. The whole instrument can be securely clamped to a table. The method of using this instrument for freezing is as follows:—The plug is first unscrewed and oiled, so as to prevent its fixture during the process of freezing. The tissue to be frozen and cut is first immersed in a thick solution of gum for some hours, "in order that the gum may permeate every part of the tissue, and prevent the formation of a crystalline condition within the frozen tissue." If the tissue has been previously immersed in alcohol, all traces of the latter should be first removed by soaking in water. Equal parts of *finely powdered* ice and salt are now placed in the freezing-box and stirred around the well, whilst the latter is filled with the solution of gum. As the gum freezes around the periphery, the tissue is plunged into it, and held until it is fixed by the advancing ice. Care should be taken to keep the exit tube of the box open, so as to allow of free escape of water, and to close the freezing box by a weighted strip of cork, to prevent the entrance of heat and to exclude the salt of the freezing mixture. By this method the freezing process may be accomplished in from ten to twenty minutes. All delicate tissues require a *special preparation* to adapt them for this process, otherwise the water which they contain freezes into hard icy spicules, which tear the structure when it is cut. Dr. Pritchard has therefore recommended a prior immersion of the tissue in thick solution of gum-arabic, using the same solution as the imbedding material.

Quite recently, Dr. Hamilton writes as follows with regard to freezing the nervous structures, and especially brain, by means of the ice-and-salt microtome. "It was found that the crystals of ice so broke up the delicate nervous tissue as to render it totally useless for minute examination. . . . I attempted two years ago to modify the method of freezing, but without success, and accordingly gave it up as an almost hopeless task."[1]

According to this statement, the writer's experience tallies wholly with my own; and fully acquainted as he was with the methods of steeping tissues in gum prior to freezing which were adopted by Dr. Rutherford, it is clear that this method

[1] 'Journal of Anat. and Phys.' vol. xii. page 257.

of freezing was not applicable for fresh brain, although it answers admirably in the case of firmer and less delicate textures. Dr. Hamilton overcame the difficulty of freezing brain with the ice-and-salt mixture, but at the expense of sacrificing the fresh for the chrome-hardening processes, a special procedure being requisite, which will be considered further on. Now it is after such considerations that we recognise the undoubted superiority of the ether-freezing microtome for nervous structures. It has been stated that the ether-freezing process has been entirely superseded by the introduction of ice and salt, or other mixtures, which by *constant* refrigeration keep the mass in a frozen state for hours. All practical manipulators, however, know that this is, as regards nervous structures, a fallacy and a blunder, since the very excellence of the ether-freezing method depends upon the fact that it can be checked at any stage and renewed when required. This is the all-important consideration in freezing brain, for beyond certain limits it is frozen into a hard icy solid, which at once blunts or turns the edge of the blade; but with the ether-spray this stage need never be attained, and a consistence is obtained admirably adapted for section-cutting. The student should therefore bear in mind that *the constant application of a freezing mixture to fresh brain cannot but result in failure for section-cutting;* and that *by use of the ether process the prior preparation of brain by hardening, or by immersion and saturation in mucilaginous fluids, may be wholly dispensed with.*

Williams' Freezing Microtome.[1]—In this instrument a wooden tub containing a freezing mixture of ice and salt is covered by a glass lid, the frame of which is secured by a clamp screw. An upright brass conducting-bar passes from the interior of this box through a central aperture in the lid, and to the extremity of this bar is screwed the circular brass plate which supports the tissue to be frozen. The arrangement of the knife is peculiar, since it is fixed in a triangular frame, and can be either raised or lowered by the screws which are adapted to the frame. There are three additional plates for

[1] This instrument is made by Messrs. Swift & Sons, University Street, London.

supporting the frozen object, and a brass cup for holding substances, which are fixed in cacao-butter or paraffin.

The box is filled with equal parts of pulverised ice and salt, care being taken to prevent the mixture touching and so fixing the cover. After the cover is replaced and screwed down, the substance to be cut is placed on the central brass plate, surrounded by a little solution of gum, and the apparatus is covered with baize to facilitate freezing. When frozen, raise the blade, and after the first cut across, proceed as follows:—Each end of the razor must be presented to the surface of the section, and *exactly levelled by means of each of the back screws.* If the large back screw be now turned, the blade can be lowered to any required extent; and since a complete revolution of the screw gives us a section $\frac{1}{100}$ of an inch in thickness, and the screw-head is graduated into sixths —a movement through sixty degrees gives us a section $\frac{1}{600}$ of an inch thick.

The special features of this microtome, in which it differs materially from others already described, consist in an arrangement whereby the blade and not the imbedded or frozen tissue becomes the movable part of the instrument; and the edge of the knife is only brought into contact with the substance to be cut.

Preparation of Hardened Brain.—In subjecting the brain to the agency of hardening reagents certain important conditions should always be kept in mind, as they are essential to success. The conditions are as follows:—

(*a*) The reagent should act equably upon all portions of the tissue.

(*b*) The requisite consistence must be acquired at the expense of the minimum of alteration and shrinking of tissue.

Now the first of these conditions can only be obtained by ensuring a thorough saturation of the tissue throughout, so that the fluid permeates rapidly to the central or deepest portions of the mass. It is evident that the surface of the tissue being bathed in the reagent will be more actively affected by the latter than the more distant parts within, and thus arises the danger of a too rapid hardening of the exterior, which forms a mechanical impediment to the permeation of the deeper

structures by the surrounding fluid. Always, therefore, take the precaution to ensure a *free and rapid permeation of the tissues by the fluid.*

Again, if any portion of the surface is in close contact with the side of the containing vessel, it necessarily is less affected than the surface bathed in the fluid. The mass should therefore be so suspended that it is *equably bathed all around in the reagent.*

Another important consideration is that of temperature, for under the most favourable circumstances the central or deeper structures must remain far less subject to the action of the reagent than the exterior of the mass, and are, therefore, the parts which most readily succumb to putrefactive changes. The larger the mass, therefore, to be hardened, the more difficult is it to prevent central decay, whilst an elevated temperature induces the same result. The tissue to be hardened should therefore be of *moderate bulk* as compared with the fluid in which it is immersed, and the preparation should be kept in a cool spot, or, better still, *in an ice safe.*

The next condition we have named, and which it is equally important to secure, is that a minimum of shrinking of tissue should result from the action of the reagent, and it is a well-known fact that all the reagents used for this purpose will, if employed in too concentrated a form, ruin the preparation by inducing extreme shrinking and brittleness of the mass; whilst again, some of these reagents are far more reliable than others, and less open to these disadvantages. The more commonly used hardening reagents are—osmic acid, Müller's fluid, solutions of the chrome salts, chromic acid, picric acid, methylated spirits, and alcohol. Now this list represents their relative value as hardening reagents for nervous tissues, osmic acid and Müller's fluid standing at the head of the series as the most valuable and least injurious in their action; chromic acid and alcohol occupying a far less prominent position in the scale. In order therefore to secure the tissue from injurious shrinking, employ in preference to the others the *reagents noted at the head of the list,* and use the *weakest solutions compatible with safety to the tissue,* commencing with the weakest, and gradually augmenting the strength of the solu-

tion, or later on, even replacing by the more astringent reagents. Let us now detail throughout the process recommended to the student for his first essay in chrome-hardening, subsequent to which it will be useful to briefly dwell upon the various modifications of the process adopted by others.

Process of Hardening by Chrome. (a) Müller's Fluid and Potassium Bichromate.—Excise a portion of the ascending frontal or parietal convolution of human brain, cutting across its length so as to remove about an inch of the convolution along with its central and deeper medulla. Lightly cover it all round with a little cotton-wool, and immerse it in from two to three ounces of methylated spirits contained in a four-ounce stoppered bottle.

Label the bottle with the name of the specimen and date, placing it in a cool cellar or ice safe.

In twenty-four hours pour away the spirit, replacing it by four ounces of Müller's fluid, the preparation being surrounded as before by cotton-wool to ensure it being bathed upon all sides alike by the fluid.

Let the bottle stand in a cool spot, and in three days replace the fluid by a fresh quantity. At the end of one week the fluid should be again renewed, or, preferably, a weak solution of potassium bichromate substituted (2 per cent.) At the end of the second week a solution of the latter of double the strength may be added; and if at the termination of the third week the mass is still pliable, and of the consistence of ordinary rubber, it is as yet unfit for section-cutting, and the reagent should be replaced by a solution of chromic acid.

In these later stages the chromic acid expedites the process without producing the extreme shrinking of tissue which ensues if it be used at earlier stages. For the same reason even absolute alcohol may at this stage be employed, but although valuable in the preparation of the spinal cord, it cannot be equally well recommended for the cortex of the hemispheres. By the above process our preparation will have attained the requisite consistence within a period of from four to eight weeks. It will be observed that in the various steps of this process the conditions previously emphasised as essential to success are obtained. Thus the early immersion in methylated spirit

abstracts all superfluous fluid from the brain and its vessels, and entering its substance by its affinity for water, aids in the rapid permeation of the mass by the chromic solution in the next stage. The more prolonged action of the spirit would, however, prove highly detrimental. Again, the cotton-wool ensures an equable distribution of fluid around, whilst the comparative bulk of the mass and fluid in which it is immersed, and the temperature to which it is exposed, provide for equable hardening to its deepest structures, and ensure it against decomposition. The gradual increase in the potency of our reagents from Müller's fluid to the strong solutions of chrome are also adjuncts to the hardening of the central portions, whilst we also expedite the process of hardening.

(*b*) *Potassium Bichromate and Chromic Acid* (Rutherford).—Place small portions of the cerebrum in methylated spirit for twenty-four hours, observing the same precautions as to relative bulk of the preparation and the reagent, covering with cotton-wool, and leaving in a cool place. Replace the spirit by a mixture of potassium bichromate and chromic acid. The proportions may advantageously be varied, according to the condition of the structure to be hardened; but the solution recommended by Rutherford, and which answers well, contains 1 gramme of chromic acid, 2 grammes of potassium bichromate to 1200 C.C. of water. Change at the end of eighteen hours, and then once a week. Should the tissue not be sufficiently tough for cutting at the end of six weeks, place it in a $\frac{1}{6}$ per cent. solution of chromic acid for a fortnight, and then in rectified spirit.

(*c*) *Iodised Spirit and Potassium Bichromate* (Betz).—Large portions of cerebrum may also be placed for a few hours in methylated spirit, tinted of a light sherry-brown by tincture of iodine. Add fresh iodine solution as the colour fades. In one or two days remove the pia mater, and return the preparation to the solution, adding to the latter half its bulk of fresh iodised spirit. After the lapse of another period of two days, replace the solution by iodised alcohol (alcohol 70°–80°) tinted of a sherry-brown by tincture of iodine). In from two to three days it should be transferred to a 4 per cent. solution of potassium bichro-

mate until sufficiently hardened for section-cutting. Should a brown deposit form over its surface during the process, let it be well washed, and a fresh solution used. When hardened, these preparations may be kept permanently in ·5 per cent. solution of bichromate. If the cerebellum is to be hardened, the segments are at once placed in the iodised alcohol, adding fresh iodine frequently as the colour of the solution pales. Remove the pia mater on the second or third day, and in a week transfer to pure methylated spirit for twenty-four hours, and finally harden it in a ·5 per cent. solution of potassium bichromate. This process, recommended by Prof. Betz, is not suitable for examination of the cortex, as the iodised spirit is injurious to the after-processes of staining. It is, however, especially suited for obtaining large sections through the hemisphere, the whole cerebrum and cerebellum, when sliced evenly across into segments ¾ of an inch thick, being most satisfactorily hardened throughout. For the minute examination of the cortex methods (*a*) and (*b*) are pre-eminently to be preferred.

(*d*) *Müller's Fluid and Ammonium Bichromate* (Hamilton).—This method is especially applicable to large segments of the brain, and is much to be preferred to the process by chromic acid (*e*). The brain is sliced completely through into segments about one inch thick. Each segment may now be placed in a large vessel, such as a brain-preparation jar, padded with cotton-wool, and containing a comparatively large bulk of the solution, which consists of three parts of Müller's fluid to one part of methylated spirit. A refrigerator or ice safe should invariably be employed to preclude decomposition, and the pieces should be turned over occasionally in the solution. In about three weeks' time they may be transferred to a solution of ammonium bichromate (·25 per cent.). At the end of the fourth week replace by a 1 per cent. solution of the same salt, and the following week by a 2 per cent. solution, in which they remain until fit for section-cutting.

(*e*.) *Chromic Acid Solution* (Lockhart Clarke).—The convolutions of the cerebrum and cerebellum were by this process hardened in a · 25 per cent. solution of the crystallised chromic acid—a stronger solution rendering them far too brittle for

section-cutting. As stated above, chromic acid is not adapted for hardening so satisfactorily and uniformly as the chrome salts. This method, employed by Lockhart Clarke, has been superseded by more reliable methods, as are also the processes recommended by Stilling, Kolliker, Hanover and Van-der-Kolk, for the hardening of nervous textures.

Hardening by Osmic Acid (Exner).[1]—It has been already stated that osmic acid (OsO_4) is one of the most reliable agents for hardening the brain and other nervous textures which we possess. Prof. Sigm. Exner of Vienna has therefore devised a method whereby *small* portions of the brain may be prepared for section-cutting by means of this reagent.

A small portion of brain, not exceeding one cubic centimetre in size, is placed in ten times its volume of a solution of osmic acid (1 per cent.). The solution should be replaced by fresh reagent of the same strength after the lapse of two days—a proceeding which may be advantageously repeated at the end of the fourth day. In from five to ten days the piece is usually stained throughout, for this reagent has the valuable property of hardening and staining simultaneously. The hardened brain is then washed in water, plunged for a second in alcohol to facilitate the imbedding, and sections are cut in the usual way in an ordinary microtome. The subsequent treatment of these sections will be given later on, when methods of staining and mounting are considered. It is only necessary here to remind the student that osmic acid is really a most valuable agent in the investigation of brain-structure, and he should avail himself of every opportunity of becoming familiar with its action.

Summary of Processes for Hardening.—The student having been thus placed in possession of the more valuable methods for preparing the brain for the imbedding microtome, it will be well to indicate the method he should adopt in his earlier attempts, as each process has its own individual merits. It is advisable that his first attempts should be made with comparatively small portions of tissue—about 3 C.C. in bulk—and that he should employ the process of hardening by Müller's fluid and potassium bichromate.

[1] Zur Kenntniss vom feineren Baue der Grosshirnrinde. Aus dem lxxxiii. Bande der Sitzb. der k. Akad. der Wissensch. iii. Abth. 1881.

(*a.*) This process, to which I assign the first place amongst the chrome-hardening processes, is slow but very certain. The results are, to my mind, more satisfactory than those of any of the other methods, and I invariably adopt it myself as the one for general use at the West Riding Asylum. The vessels which the student will find most convenient to use for hardening these smaller portions of tissue are the stoppered-glass bottles of four-ounce capacity used for dispensing purposes. Each bottle should be labelled with the name of specimen and date and nature of each successive change of reagent, whilst the specimen should occasionally be removed, and its consistence noted so as to familiarise the touch with the increasing firmness of the tissue, and the degree of hardness requisite. The method recommended by Rutherford (*b*) is also very reliable for moderate-size specimens. The methods advocated by Betz and Hamilton (*c*, *d*) are peculiarly well adapted for large segments of the brain, a whole hemisphere being thus readily hardened throughout. When these large masses of tissue have to be dealt with, special precautions are requisite. Thus the hemisphere should be sliced horizontally or vertically into segments not over $\frac{3}{4}$ of an inch in thickness. Each segment should rest upon a bed of cotton-wool in the vessel for its reception; and if two segments are included, another stratum of cotton wool should be interposed betwixt them. The vessels containing the specimen should be of large size, varying in capacity with the bulk of the latter. Thus a large preparation glass, such as is used for preserving brain in spirit on museum-shelves, will be found well adapted for hardening the whole hemisphere; whilst pickle-bottles, especially those provided with the "patent lever stopper," are most suitable for the basal ganglia, cerebellum, smaller portions of the cerebrum and the pons. Still smaller segments, such as the student will have more frequently to deal with, are best hardened in the four-ounce stoppered bottle, a good supply of which should be kept on hand. All the foregoing processes are contrasted with that of Exner's (osmic acid) in being most adapted for the demonstration of the nerve-cells and plexuses of the grey matter of the brain and its ganglia; whilst the latter, failing in this respect, is, on the other hand,

infinitely better suited for exhibiting the structure of the cerebral medulla and its extensions into the cortex of the brain. The student is therefore recommended to make himself acquainted with the structures of the cortex by the chrome methods, and subsequently to employ Exner's method for demonstrating the medullated tracts of the cortex.

Imbedding and Section-Cutting. — Having satisfied himself that the specimen is sufficiently hard for section-cutting, the student first cuts off a portion which can be accommodated by the well of the microtome, and prepares it for imbedding by a prior immersion for a few minutes in rectified spirits. This removes the fluid derived from the chrome solution from which it has been taken. In the meantime the imbedding mass should be melted at the lowest temperature requisite. The microtome-screw should be lowered sufficiently to allow of a deep imbedding of the tissue. The preparation is next removed from the spirit, rapidly dried by a fold of blotting-paper, and momentarily plunged into the warm imbedding mixture which upon its removal leaves a film over its surface, whilst all small cavities or lacuna which it may contain are filled up. The melted mass is now poured into the well of the microtome, and the preparation immersed and held in the position required for section-cutting until fixed by the cooled and consolidated mass. If the imbedding mass is one subject to much contraction on cooling, it is requisite, just as it is becoming "set," to press down with the end of a spatula the margin of the wax and oil mass against the sides of the well.

Imbedding Mixtures. — In the process of imbedding, the principle to be remembered is that the mass should be not too resistant to the blade, whilst at the same time it affords efficient support for the imbedded tissue. If the texture be one readily permeated by the imbedding mixture, contraction of the latter in cooling is apt to result in injury to the preparation, more especially if this be one of the more delicate structures. Thus in the delicate embryos of the fowl and other similar organisms, special methods of imbedding will be requisite. For the brain and spinal cord, however, the process is comparatively simple, since

the imbedding mass neither permeates its structures nor injures it by contracting.

The following are the more important media employed for imbedding prior to section-cutting:

1. White wax. Olive oil, equal parts.
2. White wax, 3 parts. Olive oil, 1 part.
3. White wax. Cacao-butter.
4. Solid paraffin, 5 parts. Hog's lard, 1 part.
5. Solid paraffin, 5 parts. Paraffin oil, 1 part. Hog's lard, 1 part.
6. Solid paraffin, 5 parts. Spermaceti, 2 parts. Hog's lard, 1 part.
7. Spermaceti, 4 parts. Cacao-butter, 1 part.
8. Spermaceti, 4 parts. Castor oil, 1 part.

The first on this list is perhaps the more generally employed medium, but it possesses the disadvantage of contracting strongly on cooling, so as to leave an interval betwixt the wax plug and the sides of the well: this, of course, will necessarily tend to loosen the mass, and allow rotatory or wobbling movements, which prevent accurate section-cutting. These disadvantages may be overcome by one or other of the following measures:

Vary the proportion of the oil and wax in favour of the former. When solidifying, press down the edge of the mass with a spatula. Use a microtome plug with groove or projections on upper surface. Drive the mass out of the well, partly surround it with a narrow strip of blotting-paper and return it—forcibly pressing it down into the well—the blotting-paper swells by imbibition of the spirit in section-cutting, and so fixes the plug firmly.

In the use of the third medium, viz. white wax and cacao-butter, far less contraction ensues upon solidification, and a firm supporting mass is obtained, the proportion of the ingredients varying with the firmness required. I can also speak favourably of the combination of paraffin with hog's lard. In all these cases it is well to keep the mass in a small tinned pot with lip, provided with cover and handle, so that it can readily be held over a gas-jet or Bunsen burner, and melted as required. Another imbedding agent used is gum, which is solidified by the application of alcohol or

methylated spirits. This method, proposed by Brücke, although valuable for many tissues, cannot be recommended for brain.[1]

Imbedding is wholly dispensed with in the form of microtome devised by Schiefferdecker, where the preparation having been hardened by alcohol as far as practicable, is placed in the well and clamped firmly by the arrangement already described. In the small hand-microtome of Ranvier, again, the ordinary melted media for imbedding are often exchanged for elder-pith, which is packed around the tissue in the well in a dry state, and then, upon immersion in spirit, the pith swells and firmly fixes the preparation. None of these latter methods are so suitable for hardened brain as imbedding in the wax and paraffin mixtures.

Section-Cutting.—The preparation being satisfactorily imbedded, and the mass perfectly cold and hard, our next procedure is to cut the *finest possible sections—they cannot be cut too fine.* Place on the table supporting the microtome, and in front of the latter, two vessels, one a Griffin beaker-glass of three inches diameter, and the other a cylindrical glass jar, a brain-preparation jar, or better still, a flat-bottomed porcelain evaporating basin. Nearly fill the beaker with methylated spirit, and the latter with water. Seat yourself in front of the microtome at a convenient level for the free play of the hands, place a soft towel over the knees, to be used for wiping off adherent wax from the imbedding mass. Have close at hand, also, a razor-strop, a porcelain capsule of small size, and some camel-hair brushes.

Dip the section-blade in the methylated spirit, and having raised the imbedded mass slightly above the level of the section-plate, slice off the superficial portion, exposing a clean smooth surface of the tissue for cutting. Again dip the blade into the spirit, and turning the microtome-screw, say through one-eighth of a revolution, place the flat of the blade upon the section-plate in front of the mass, with the back towards yourself and the handle in your right hand, grasped near the heel of the blade. Now cut your first section by a clean steady sweep from heel to point, and always *away* from your-

[1] 'Handbook of the Physiological Laboratory,' page 92.

self. No saw-like movement should be performed, nor should the blade be arrested occasionally in its course, the section shaved continuously and evenly off the preparation. The thin film of wax removed from the front of the tissue is sometimes likely to get in the way of very fine sections, and hence it is usually advisable, with a penknife to gently remove all the imbedding mass *in front* of the preparation to about the depth of a couple of millemetres prior to section-cutting. Subsequent sections should be cut with a still more restricted movement of the screw, until the operator has found the minimum degree of movement at which a section can be cut. This, of course, is a matter of tact, and the student will find that he daily improves the quality of his sections by practical manipulation. Two precautions must ever be taken if successful sections are desired. The first is, that as the blade is raised out of the spirit, the latter should not be drained off, but a good flow be retained over the surface of the blade, so that the section is floated up as it is cut. In the next place make it a rule to pass the blade across the strop frequently, after cutting half-a-dozen sections, as it requires to be kept at the finest possible edge. As the sections are cut they are floated off into the porcelain basin of water, and are freed from adherent particles of the imbedding mass by the rapid gyratory movements they here undergo from the currents caused by the attraction of water for the spirit they contain. After floating off each section in the water, pass the blade softly over the towel to clear its surface from adherent wax, prior to bathing it afresh in the spirit. How is the student to ascertain whether his sections be sufficiently thin? The exact thickness of the section is ascertained if the pitch of the microtome screw is known, and the degree of movement which he adopts noted. Thus, if the screw have eighty threads to the inch, a movement through one-eighth of a complete turn, i.e. 45°, will give a section $\frac{1}{640}$th of an inch in thickness, whilst a movement through an arc of 30°, or $\frac{1}{12}$th the entire turn, gives a section $\frac{1}{960}$th of an inch thick. He will, however, be not far from wrong by following this rule: reject all sections which do not float, and of the latter preserve as valuable only those which appear as a mere film on the surface

of the fluid, requiring to be looked at sideways to be distinctly seen. It is at this stage of the proceeding, that of section-cutting, that we can best judge of the merits of the imbedding mass, which, if found unnecessarily resistant to the blade, should be modified by the addition of more of the diluting adjuncts, viz., lard, oil, or cacao-butter.

The Section Blade.—This is an item of supreme importance to the histologist, and it is by no means true, as we often find asserted, that any razor blade answers the purpose equally well with the specially prepared knives. I have found this statement made frequently by those who were by no means fortunate in the thinness and delicacy of their sections; whilst all whom I am acquainted with, who can rejoice in the good qualities of their sections, have always paid great attention to the section-blade which they employ. The qualities of a good blade for section-cutting upon the microtome depend upon its make, form, edge, and tempering. It should be sufficiently long to ensure a free sweep over the whole surface of the section plate from heel to point, wide enough from back to edge to support and float up the largest-sized section which might have to be cut; it should be hollowed out on both surfaces, but be most concave on the surface uppermost in section-cutting; the edge should be extremely sharp and perfect, whilst the angle formed by the converging surfaces here should not be so acute as to involve bending of the blade when subjected to slight pressure; the quality and tempering of the steel should be of the best character. The above points are all most essential requirements in a good section-blade; but another important consideration is that the plane of the back and edge of the knife should exactly coincide when it rests upon a perfectly level surface as the microtome section-plate—not the slightest degree of tilting being admissible.

For the smaller microtomes I have invariably used razor-blades, my three instruments measuring in length and width of cutting-surface as follows:—

No. 1.	4 Inches	×	1 Inch.	(made by Weedon).
	$4\frac{1}{2}$ „	×	1 „	(„ by Young).
	5 „	×	$1\frac{1}{4}$ „	(„ by Young).

The first is a most valuable knife. It is fitted into a short,

firm, and fixed handle. The latter, which is my favourite blade, has a folding handle, and is sketched in outline with a section of its concave surfaces in an early number of 'BRAIN.'[1] Some of the earliest and most discouraging failures upon the part of the tyro in section-cutting depend upon the condition of the blade, and it is necessary that he should be fully impressed with the fact that not only must he secure a couple of section-knives with the above qualities, but that he should jealously keep them in most perfect order. To ensure the latter condition, let him observe the following precautions:—

Use no undue pressure against the section-plate.

Keep the surface of the blade absolutely level with the plate. Pass it frequently along the strop whilst cutting sections. After each day's work examine its edge most critically, and if the slightest irregularity or notching be apparent it must be reset before use.

Keep the blade dry and well polished by a soft handkerchief, and if in a closed handle, have a chamois-leather case made for it.

Subsequent treatment of Sections.—The sections have now to be removed carefully by a camel-hair brush to methylated spirit contained in the small porcelain capsule, in which they may be allowed to soak for some time to remove all traces of chrome, which otherwise interferes with the subsequent staining they have to undergo.

Large Sections through Hemisphere.—The above account refers especially to the smaller class of sections. When it is requisite to obtain sections upon a much larger scale, such as those through the hemisphere or the whole brain, the process requires modification. The method of imbedding is precisely similar in all its details, but when we come to cutting the section, special forms of knife are found requisite. The blade which I use measures 16 inches long by 2 inches wide, and is fixed in upright handles at each end. The zinc basin in which the large microtome rests is filled with water, and then sections are cut with a saw-like movement of the blade, and not, as in the former cases, by one complete sweep through the structures. It requires a steady hand and much manipulative skill to

[1] Vide 'BRAIN,' Part III. page 353. October, 1878.

obtain these larger sections both thin and perfect, and the student will only perfect himself by frequent and persevering efforts. These large sections can be readily removed by means of sheets of paper upon which they are floated, and this support may be given to them throughout the various subsequent stages of preparation. When the desired number of sections have been obtained, the imbedding plug may be removed, the remaining portion of tissue placed in methylated spirit, or a 5 per cent. solution of bichromate of potash, until required. The microtome should be carefully dried and placed away in a drawer for safety, as the slightest scratching of the polished section-plate will ruin a good blade in a few minutes.

The next stage through which our sections pass is that of staining and mounting, and this we had best leave for consideration in our next article.

ON THE BLENDING OF COLOURS BY THE SOLE AGENCY OF THE SENSORIUM.

BY JOHN GORHAM, M.R.C.S., TUNBRIDGE.

In order to obtain single vision with the two eyes, the images are known to impinge on identical or corresponding parts of the two retinæ; if these parts do not coincide, there is double vision. But the two eyes may be made to perform separate functions; the one to receive a colour-sensation, the other to discharge it.

There has been a good deal of controversy as to whether a true blending or mixture of two colours actually does take place in the brain.

The subjoined experiments show that it is possible to obtain a conduction or propagation of white or coloured sensation from one eye to the other, so that if a red light is presented to the right eye and a green to the left, the two sensations shall be united in the brain and call up that of yellow.

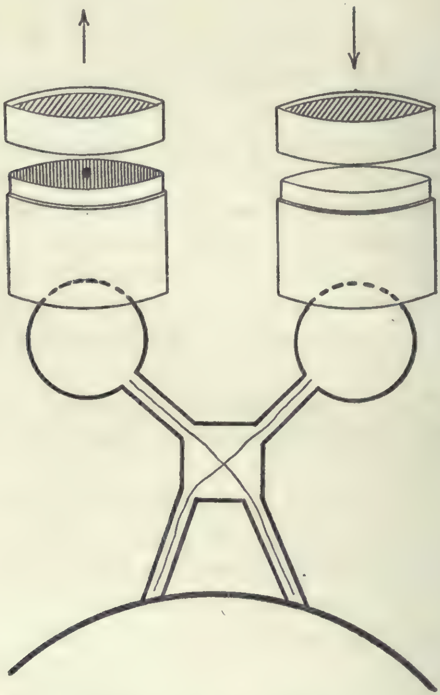
Such combinations I have found to take place when a large quantity of white or coloured light is caused to impinge on the retina of one eye, while a very small quantity falls on that of the opposite eye.

The only apparatus required is a couple of ounce boxes, which are converted into tubes by removing their lids and pushing their bottoms out; and four pairs of lids.

The tube used for the right eye is left open at both ends (diagram), and coated inside with lamp-black; that for the left eye is closed at one end by a thin metallic disc, perforated at its centre with a small needle the one-fiftieth of an inch in diameter. It is then blackened inside. The lids, having their tops removed, are mounted with coloured gelatine films, four

films to each lid; this forms a fair substitute for coloured glass, and is easily procured. Eight lids are used, and they are mounted in pairs, with green, blue, red, and yellow gelatine.

To simplify description, we will assume that the open tube is always applied to the right eye, the punctured one to the left.



EXPLANATION OF THE DIAGRAM.

The diagram shows the two lids mounted with discs of coloured gelatine, and the tubes through which light is admitted to the eyes. That for the right eye (to the right hand) is open at both ends, to admit much light; the tube for the left eye is closed at one end, and punctured with a fine needle, to admit of little light. Below are shown the two eyes engaged in looking through their respective tubes, with their optic nerves approximated in a square space (optic commissure) and having cross fibres which carry nervous energy from the eyes to opposite portions of the brain. The thick curve line at the bottom denotes a portion of the brain, from which these nerves derive their origin.

By this peculiar arrangement, light which is admitted to the right eye is found by experiment to take a circuit through the optic nerves and brain, finding its way to the opposite eye, and displaying itself in the disc made by the needle. This accomplished, the problem of *binocular* mixture is solved.

By this arrangement we impress both eyes with white or coloured light, the same in *purity*, similar or dissimilar in *hue*, but totally different in *degree*. The right eye is saturated with much light through a large orifice, the left with little light through a minute needle-hole.

The object of using tubes is to ensure that each eye shall be impressed with its own light unmixed with that which enters the other.

We first present a colour to the right eye, and white light to the left.

Closing the right tube by a lid of green gelatine, and admitting white light to the left eye through the needle-hole, we direct our gaze on a sheet of white paper spread open on a table.

Each eye has now become impressed with an image of its own; that seen by the right eye is a large round *green* disc, that by the left eye a small round *pink* one.

What is the cause of the pink colour of this little disc? that it is connected in some way with the sensation engaging the opposite eye there can be no doubt, for on shutting that eye the pink appearance of the little disc has vanished and is replaced by white light. On re-opening the right eye the white colour of the little disc is exchanged for pink, and this alternation of the two colours takes place as often as we choose to repeat the trial.

But our experiment is only half-completed; for when the two eyes are fully impressed with their colour respectively, if we now suddenly remove the green from the right eye the left disc loses its pink hue and becomes green.

Here, then, we have conclusive evidence from direct experiment that, when one eye is impressed with a colour-sensation, the other eye responds and participates in the effect; and, moreover, that this has not resulted from any external source, but from direct communication from eye to eye within the cranium through the brain itself. Corroborating evidence is obtained by trials with other colours.

We now cover the tube of the right eye with a lid of *blue*. Directing our gaze on a white surface, as before, we see a large *blue* disc, a smaller *yellow* one. On suddenly removing the

blue tube, keeping the right eye still open, the small yellow disc has as suddenly become blue.

Again: the right eye was similarly impressed with a *yellow* colour; its complementary *blue* was quickly imparted to the smaller disc seen by the left eye. But the simplest experiment of all is perhaps quite as conclusive as any.

We apply the tube to the left eye, and for a yellow film we substitute the yellow light emitted from a gas flame. Gazing on a white surface, with the right eye shut, the small disc is seen nearly white. We now open the right eye, when the small disc immediately assumes a beautiful cobalt-blue appearance, evidently the complementary of the light impinging on the opposite retina.

We arrive at the conclusion, therefore, that *when one retina is fully impressed with the sensation of a given colour, and the other retina feebly illuminated with white light, a current of colour is set up between the two eyes, and that this current is propagated from that eye which receives the most light towards that which receives the least. And, moreover, that which is transmitted to the left eye is first the complementary, and then the colour itself, of that which impinges on the right retina.*

On Sensorial Mixture when a different Colour is presented to the Right and Left Eye.—We place a lid of *green* film before the puncture in the left tube, and a lid of *red* film before the right eye.

Bearing in mind our former experiments, and utilising them in this, we are able to effect a combination of these two colours, just as when they are rotated on a wheel and form a yellow light.

Holding both tubes close to the eyes, and looking, as before, at a sheet of white paper, we observe first that the green of the small disc is intensified. This we anticipated; it has received the complement evoked from the red—we see, so to speak, green plus green. We now suddenly remove the red film from the right eye; the little green disc has changed to *yellow*. This, again, was confidently expected, for the original colour (*red*) being suddenly withdrawn from the right eye, it is then, and then only, that it appears to the opposite eye. It is at this juncture, therefore, that the two sensations really blend and form the binary *yellow* hue.

We do not always succeed upon a merely first trial, for the right eye often requires replenishing by a frequent application and quick withdrawal of the red tube. By this process of "feeding" the one eye, as Bacon has it, the little disc of the other gradually participates, loses its green appearance, and emerges finally into a pure yellow.

And if we reverse the position of the colours, placing the green before the right eye and the red before the left, the two blend as before, and form a brilliant yellow.

The yellow thus procured is as pure and well-mixed as that which is formed in one eye by the rotation on a wheel of equal sections of red and green. It contrasts strikingly with that kind of binocular mixture which has been attempted by the aid of the stereoscope, but with signal failure; the colours so nearly blending without actually doing so, that the appearance is most tantalising, while the individuality of each is never entirely lost.

To combine "Red" and "Blue."—We use a lid of *blue* gelatine for the left eye, and one of *red* for the right eye. Gazing on a white surface, we now observe a large red disc and a smaller blue one. This little blue disc is slightly deteriorated by receiving a green complementary from the opposite side.

We now suddenly remove the right tube and examine the effect this has produced on the blue disc. On repeating this process, say a dozen times, the blue has gradually become charged with red, and passes through a whole series of violet hues, from blue-violet to red-violet.

We have seen that in order to convert this blue disc into one of violet it must be made to combine with red, and also that this red sensation must, in some way, be procured from the right eye. Still the mode by which this is accomplished is remarkable, for it can be effected only by actually depriving the right eye at brief intervals with the very colour which impresses it, and by no other means. Hence, on withdrawing the red tube, its colour directly finds its way across to tinge the blue, and, by repeating the process, the blue gradually becomes charged with sufficient red to assume a violet hue. In this way we "feed," so to speak, the left eye with red, by

repeatedly withdrawing this colour which actually supplies it from the right eye.

To combine "Blue" and "Yellow."—These colours are complementary, and, when blended, form white light.

We apply a tube, armed with *blue*, to the left eye, and a *yellow* film to the right eye. Directing our gaze on a white surface, we see a large yellow disc, a small blue one.

The blue appearance of the little disc is seen to be enhanced by receiving the complementary of yellow from the opposite eye.

We now remove the yellow tube from the right eye. The effect of this is directly perceived; a propagation of yellow light takes place from right to left, and after a few repetitions the blue disc is completely merged in white.

To multiply experiments would be irksome to my readers, and encroaching on the pages of this Journal. It may be interesting to remark, in conclusion, that the complementaries, one and all, may be caused to blend, forming white light by impressing each eye with the *same* colour, when that of the little disc immediately merges into white. This result is inevitable indeed, and tends to confirm the legitimacy of the plan now proposed for showing that a true blending or mixture of two colours actually does take place in the brain, and that the mixture itself is always located, and may be perceived in a little disc prepared expressly for the purpose.

By the same arrangement as that shown in diagram, *motor* force, as well as *colour*-sensation, is found to be communicable from eye to eye, causing contraction and dilatation of the pupil. An account of this is reserved for a future communication.

A CLINICAL LECTURE ON SHAKING PALSY.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

THE disease "Shaking Palsy," or "Paralysis agitans," some examples of which I shall have the opportunity of showing you, was first regularly described by our countryman Parkinson in 1817. Parkinson was a member of the Royal College of Surgeons, and his 'Essay on the Shaking Palsy' presents so graphic and admirable a description of the disease, that comparatively little has been left for subsequent observers to add to his account. In our time Charcot has also made the disease the subject of clinical investigation. The influence of these writers cannot fail to be felt at every turn by any one desirous of demonstrating the features of this remarkable affection. Parkinson's definition of Paralysis agitans is as follows:

"Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards and to pass from a walking to a running pace: the senses and intellect being uninjured."

He quotes the distinction between kinds of tremor which was drawn by Sylvius de la Boe, who contrasts tremors produced by attempts at voluntary motion with those which occur whilst the body is at rest. Sauvages in 1763 had distinguished the latter species (Tremor coactus) by observing that "the tremulous parts leap and as it were vibrate even when supported;" whilst every other tremor, he observes, ceases when the voluntary exertion for moving the limb stops, or the part is supported, but returns when we will the limb to move.

We still depend very much upon this point—the difference of circumstances in which the tremor shows itself, for our diagnosis of Paralysis agitans.

The female patient, Mrs. G., is a widow, æt. 62. She sits with her chin resting on her chest, the lower lip and the neighbouring muscular structures about the chin being constantly in tremulous movement. There is a frown upon her brows. Her hands lie in her lap, the fingers being somewhat interlaced, apparently for the comfort of mutual support. When separated, the hands chiefly, and the forearms to a less extent, are in a state of constant tremor. If we count the to-and-fro movements, we find them to number from 160–170 in the minute. The tremor seems to be about equal in each hand.

When I speak to her and she answers, the shaking of the hands becomes more marked. It is not that the rapidity, however, is increased, but the movements occupy a larger space. My hand placed upon her knees cannot distinguish any trembling of the legs.

She can stand and walk, as you see, without help, but with short, "toddling" steps, the head being carried low and the body stooping forward. The face shows little or no expression, and wears a kind of fixed look. This attitude and general bearing it is very important to note carefully; they are characteristic of the disease. As she stands with her back towards me, if I pull her by the dress, with even the slightest force, she tends to fall backwards. When asked to lay hold of a cup which is offered to her, she puts out her right hand to take it, and whilst doing so the movements entirely cease. When, however, she has got hold of it and is told to squeeze the handle the movements return.

In another form of shaking palsy, that dependent upon insular, or multilocular, or disseminated sclerosis, as it is variously called, "the tremor," as Charcot has pointed out, "only manifests itself on the occasion of intentional movements of some extent; it ceases to exist when the muscles are abandoned to complete repose." The cessation of tremor in this woman when she stretches out her hand to take the cup is not an exceptional circumstance. In many cases of Paralysis agitans, it is true,

the tremor will persist throughout the voluntary movement. But the present feature also is common enough, and has not escaped the observation of Parkinson, who contrasts it with what happens in that which he styles "tremor." "It is necessary to bear in mind," he writes, "that this affection" (shaking palsy) "is distinguishable from tremor by the agitation in the former occurring whilst the affected part is supported and unemployed, *and being even checked by the adoption of voluntary motion;* whilst in the latter the tremor is induced immediately on bringing the parts into action. Thus an artist afflicted with the malady here treated of, whilst his hand and arm are palpitating strongly, will seize his pencil, and the motions will be suspended, allowing him to use it for a short period; but in tremor, if the hand be quite free from the affection, should the pen or pencil be taken up the trembling immediately commences."

Charcot, also, when referring to cases of shaking palsy in which the tremor only occurs intermittently, says: "Singularly enough, it is just in such cases that the tremor shows itself rather when the limbs are at rest, and ceases when they are set in motion by the will."

There can be no doubt that in general the persistence of the movements of the hands when the muscles are abandoned to repose, is sufficient to differentiate Paralysis agitans from insular sclerosis. This is a rule, however, which is by no means without exceptions. In one of my cases, which an autopsy showed was an example of insular sclerosis, the tremor occurred as well in a condition of repose as on the occasion of intentional movement; on the other hand, I think I can call to mind more than one case belonging probably to the class of Paralysis agitans in which the tremor was disposed to cease during muscular repose, and evince itself during voluntary movement of the limb.

The attitude of this patient's hands is another very noteworthy feature, and I would ask you to remember it particularly in reference to an examination we shall presently make of the other patient. It is, as Charcot has shown, very much the attitude of the writing hand, the fingers being inclined to an angle of about 45° with the metacarpal portion of the

hand. You will note, however, that the fingers do not present the easy curve seen in the writing hand. Their position, it seems to me, is precisely that which is obtained by faradising the dorsal interossii—the first phalanx is flexed, the second and third being extended upon the first. The tremulous movements of the thumb laterally, as well as backwards and forwards, are marked more strongly than those of the fingers, and convey the idea of the patient rolling some object between it and the fingers. The fingers, *en masse*, tend to deviate towards the ulnar side. The extended position of the fingers would seem to indicate a tendency to muscular rigidity as well as convulsion, and this is indeed what we find when we come to examine other parts of the body. It is especially well seen in the fixed position of the head and neck; the chin can be lifted, but it soon resumes its position. We also learn from the patient that she has a great tendency to become cramped in her limbs. This rigidity, as we shall see in the other patient, may become the most dominant feature of the case. When we take an aggregation of symptoms—*Symptomen-complex*, I think the Germans call it—and give a name to the disease which is characterised by the association of these symptoms, we must take care to remember that in various examples individual symptoms will certainly vary in their prominence. It will often happen that a symptom which perhaps we have thought of but lightly—have "thrown in" among the rest, as it were—will in a certain case assert itself so strongly as to dwarf those others, upon the presence of which we are accustomed to rely for making a diagnosis. On the other hand, a symptom which we have come to look upon as essential, may be actually absent. In this woman the presence of the peculiar tremor of the hands must be allowed to be, as indeed happens in the majority of cases of Paralysis agitans, the most striking feature. I have shown you, also, the slight indication there is of a tendency to rigidity of the muscular system. In her case this is a symptom pronounced only in the most trifling degree. We can readily imagine, however, that this symptom might be the most dominant one in a certain case, and that the tremor in its turn might be absent or only slightly marked. I shall be able to show you presently a man

who, I think, exhibits this kind of anomaly. But for the present let us return to the consideration of the disease of which we have a typical example in the person of this female.

More often than not, I think, the muscles of the face fail to show, to a great extent at least, the tremor which is so marked in the hands, but in this patient there is very distinct tremulous movement of the lips, especially the under lip. And in consequence of this inability to keep the mouth sufficiently closed, the saliva is almost constantly dribbling, much as we see it in cases of glosso-labio-pharyngeal paralysis. There is no difficulty, according to her account, in swallowing.

Mrs. G. suffers much from flushes of heat, and her face is ever and anon covered with a deep blush. In many cases these symptoms are accompanied by profuse general perspiration, but this is not observed in the present instance. An "habitual sensation of excessive heat" was described, for the first time, by Charcot as characterising Paralysis agitans.

There is a symptom in this patient which, according to my experience, is a very frequent one, but which has not hitherto, so far as I know, received attention at the hands of any observer. Charcot, it is true, has described the affection of speech, which is often observed, in these words: "There is no real difficulty of speech, but the utterance is slow, jerky, and short of phrase; the pronunciation of each word appears to cost a considerable effort of the will. If the tremor of the body be intense, it may happen that the utterance may be tremulous, broken, jolted out, as it were, like that of an inexperienced rider on horseback when the animal is trotting. However, in both cases we should recognise in this a phenomenon of transmitted tremor. Finally, the patients seem to speak between their teeth." We observe something of the kind in this patient, but the symptom upon which I now wish to speak concerns not the articulation but the voice. The voice has a peculiar "piping" character. It is, if you will observe, the conventional voice of the old man upon the stage. We all know that on the stage peculiarities of every kind are obliged to be accentuated for the sake of strong light and shade. When a very advanced period of old age has to be represented, the tone of voice adopted is exactly of the shrill

piping character which we note in this woman, who, however, is only sixty-two years of age. Since I first noted this "piping" note in cases of shaking palsy many years ago, I have been interested in observing whether it is common in the exceptionally aged persons (not afflicted with Paralysis agitans) whom from time to time I have met. I have not found it to be so. Now, Paralysis agitans, although, as Charcot has pointed out, it especially assails persons who have passed their fortieth or fiftieth year, has a slow march and little or no tendency to curtail longevity. Hence there are always about a certain number of persons afflicted with Paralysis agitans who have arrived at a ripe old age, and who present this peculiarity of voice. I cannot help thinking that the conventional voice of age on the stage has originally been derived from the study of some old person affected with this disease, and thence handed down, as we know is the stage custom, to successive generations of performers.

A slight tremulousness shows itself in the patient's tongue. There is no affection of the cutaneous sensibility, nor of the special senses. The bowels are very obstinate, so that she is habitually obliged to take aperient medicine. She can retain the fæces, but has to be very quick in relieving her bladder, and requires to get up five or six times every night for the purpose. She suffers pain of an aching character in her limbs.

We do not observe in this patient a symptom which is so common in this disease as often to enable us to recognise a case at a glance. Parkinson describes it as "a propensity to bend the trunk forwards and to pass from a walking to a running pace." Although the patient presents the fixed head and stooping body, she does not hurry along as we so often see in such cases. There is no "festination," to use the term employed to designate this hurried march.

The amount of muscular power varies much in individual instances, and also in different stages of the disease. At first there may be but little notable weakness; but as time goes on, a very distinct loss of power shows itself in the limbs and especially in those affected by tremor. In this woman the power of grasp is only feeble.

Before proceeding to consider other points connected with the symptoms of this peculiarly distressing disease, let us examine the other patient whom I have brought before you. I am indebted to Dr. Beevor's notes for many particulars of his case.

James H. is 64 years of age. He has been an upholsterer's foreman, has worked hard, and, according to his own account, has always been steady. He has been married 30 years, has had four children, and his wife has never miscarried. There is no history of phthisis, insanity, or fits in his family, but a doubtful account of hemiplegia in his mother. He says that he never had scarlet fever or rheumatic fever, or syphilis. He had "typhus" fever when young, and has suffered a little from gravel. His present illness dates from only two years.

At first he found difficulty in using his knife and fork and in putting on his clothes. About four months later he began to drag his feet in walking, and gradually power was lost more and more completely in all his limbs. Six months ago he began to "speak thick," according to his own description, and this has since become worse. He has not had trouble in masticating, and could always protrude his tongue. He says that he has sometimes choked in swallowing, but no difficulty has been observed in this respect since his admission into hospital. For six months past he has noticed that when his arms were raised they remained in the acquired position longer than was natural. He has not been able to walk for a year past, or even to get out of bed. His legs and arms have been stiff for the last nine months.

The patient is a very stout old man, who constantly lies in bed without occupying himself in any way. He cannot help himself at all. There is an aspect of marked mental hebetude, or at all events an extreme slowness of expression, so that it is difficult to elicit answers to questions about his history. The difficulty is partly also attendant upon his mumbling speech, which is sometimes quite unintelligible. Yet we contrive to get, in process of time, though the task is laborious, a fair amount of information from him. The face wears a peculiarly stolid expression. When told to show his teeth he makes only a slow and feeble attempt; he can close his eyes,

but cannot screw his eyelids up tight. The tongue is protruded straight although slowly, and it does not show any tremor. Cutaneous sensibility is nowhere affected. The patient has not suffered from pains in his head or body. The movements of the eye-muscles are natural. There is no nystagmus.

The upper extremities are thin in proportion to the lower, but the emaciation, if so it can be called, is symmetrical on the two sides, as is shown by the following measurements. Right forearm, $8\frac{3}{4}$ inches; left forearm, $8\frac{1}{2}$ inches. Right arm, $9\frac{1}{2}$ inches; left arm, $9\frac{1}{4}$ inches. The hands remain in a position of semiflexion, the fourth and fifth fingers more flexed than the second and third. The finger-portion of the hand is much drawn over to the ulnar side—the thumb is applied against the forefinger. The attitude of the hands is exactly that of Paralysis agitans, but there is no tremor whatever in the fingers. The two distal phalanges appear somewhat over-extended upon the proximal phalanx. The intrinsic muscles of the thumb and the interossei appear to be thin.

As you observe, this man can flex and extend his fingers, but the movements are exceedingly slow. He can, in the same deliberate way, flex and extend (though not to the full extent) both elbow-joints, but there is very little capacity for pronation or supination. He can raise his arms straight up into the air and abduct them from his trunk, the shoulder-joints being less affected than the elbows.

There is a very curious circumstance to be noted in connection with the movements of his limbs. In response to my request, he raises his hand or his foot to some distance above his couch, and there he lets it remain for several seconds until, indeed, he is told to drop it; and if I lift one of his limbs, and place it in any position, so it remains in a cataleptic fashion. When told to replace it, it is long before he makes any movement, and then the limb descends slowly. The muscles of his trunk appear to be weak; he cannot raise himself in bed, and, indeed, does not move at all—lying, if not disturbed, for hours together in the position in which he is placed.

As regards the lower limbs, they are not so much emaciated as the upper extremities, but the muscles are flabby. He can

lift either heel very slowly off the couch, can bend either knee, but can only move the ankle-joints a very little. All the joints appear very stiff, and require considerable force to move them.

The patellar tendon-reflex is only just obtained. There is no ankle-clonus. The cutaneous reflex of the foot-sole is obtained on each side.

The right pupil is smaller than the left. They both react to light.

The heart-sounds appear distant; there is no murmur. As regards his mental state, in reply to inquiry he says that "he feels lost." He says "he wants to give his address," and gives 139 Bow Road, which is not altogether correct. He seems very obtuse, and scarcely speaks at all, though he is always apparently conscious.

The faradaic excitability of the muscles of the forearms is normal, that of the arms and lower extremities exhibits a very slight and unimportant lowering.

The action of the rectum and bladder is preserved. He has no difficulty in retaining his urine or fæces, but sometimes a little delay occurs in expelling the contents of the bladder.

There is no disposition towards the formation of bed-sores.

The ophthalmoscope shows no change in the fundus oculi.

These represent the principal symptoms of a condition which is manifestly of an unusual and obscure character. I have been led to conclude that the patient represents an anomalous form of Paralysis agitans, partly by the necessity of excluding other explanations, and partly by the resemblance of the patient's state to that which would be produced by an intensification of certain symptoms of shaking palsy. Let us first see how far his condition can be made to tally with that produced by certain other conditions.

The preserved electric irritability of the muscles enables us at once to exclude, what otherwise might be a not unlikely supposition, that the patient is suffering from a severe form of lead-poisoning. In such a condition the faradaic excitability would be very much reduced, or, still more probably, absent. Progressive muscular atrophy is excluded by the universal character of the lesion. As you are aware, that disease picks

out groups of muscles physiologically associated in their action, leaving others intact. Here the affection is a general one of the whole muscular system, and, in addition to this, the mental condition, or, at least, the laborious slowness of movement in response to order which we observe in this man, forms no part of the history of progressive muscular atrophy. Bilateral sclerosis, which at first suggests itself, on account of the rigidity of the limbs, is excluded by the condition of the tendon-reflexes, which in that disease would certainly be exaggerated. So also would they be, though not to an equal extent, in Charcot's amyotrophic lateral sclerosis. In both these conditions, the one dependent on sclerosis of the lateral columns alone, and the other upon sclerosis of those columns along with lesion of the large ganglionic cells in the anterior cornua, the tendon-reflexes are intensified. For the same reason, cerebro-spinal insular sclerosis must also be excluded.

On the other hand, let us see what symptoms are to be found which accord with the view that the patient is affected with Paralysis agitans. We have not here, it must be remembered, the opportunity of seeing him walk. That is a great loss, for the attitude of a patient with shaking palsy is, as you have seen, exceedingly characteristic. "The patient," as Charcot says, "loses the faculty of preserving equilibrium whilst walking. In some we notice a tendency to propulsion or to retropulsion;" (in this female, as I showed just now, the tendency to retropulsion when I pulled the skirt of her dress is well marked) "without feeling any giddiness, the patient is in the first case propelled forward and, as it were, compelled to adopt a quick pace; the individual is unable without extreme difficulty to stop—being apparently forced to follow a flying centre of gravity." He adds: "A peculiar attitude of the body and its members, a fixed look, and immobile features should also be enumerated among the more important symptoms of this disease." Again: "The muscles of the face are motionless, there is even a remarkable fixity of look, and the features present a permanent expression of mournfulness, sometimes of stolidness or stupidity."

Although we cannot see this patient stand or walk we have the opportunity of noting the fixed look and immobile

features which singularly resemble this description, as well as what we observe in the other patient. Might not a widespread softening of the brain be attended with a similar physiognomy? I think it might. But it must be remembered that the mode of progress of the disease in this man does not accord with such a supposition. There is a gradually progressive loss of power and increasing rigidity in his members. When you have widespread atheromatous changes in cerebral arteries leading to thrombosis and softening, the paralysis which ensues, although it may easily involve all the limbs, does so not by slow and imperceptible degrees, but by leaps. A more or less sudden loss of power occurs in one limb, and after an interval of varying duration the same thing happens in regard to another. Moreover in such a case again we should certainly expect the tendon-reflexes to be exaggerated —not lowered in activity as is the case in this man's knees. The same argument applies against the suggestion of the growth of a cerebral tumour, which besides is additionally rendered improbable, though not, it must be allowed, impossible by the absence at any time of pain in the head, and vomiting, and the fact that no changes are discernible with the ophthalmoscope. But there are still two other circumstances which, although not absolutely conclusive, speak most strongly against the view that the condition described depends on a destructive intracranial lesion. The patient is able to retain his urine and fæces. If his helplessness were dependent upon extensive brain softening, it is in the highest degree improbable that the power over the bladder and rectum would have been maintained. I mention this as a result of clinical experience without being able to say what portion of the encephalic centres must be involved to induce this particular loss of power. It is a fact that in more or less generalised paralysis from cerebral softening we do constantly find that the evacuations cannot be restrained.

The second circumstance is that the patient, though he lies in the position in which he is placed, shows no disposition to the formation of bed-sores. It is true that he was placed on a water-bed as soon as he arrived, but the helplessness which we see had existed for months before his admission into hospital,

and during that time he did not enjoy this advantage. But besides the expressionless features and fixity of looks, we have here two other symptoms of Paralysis agitans of great importance. The first is the attitude of the fingers which, as you have seen, precisely resembles that which is to be observed in the female patient. Now this attitude is very peculiar. It does not occur in ordinary cases of hemiplegia or generalised paralysis from extensive cerebral softening. In such cases it is true the hand tends to assume a position of more or less strongly marked flexion, but *the fingers are also and indeed especially flexed.* Here on the contrary the forefinger and middle finger at least tend rather towards an attitude of over-extension. The phalangeal portion of the hand is besides inclined towards the ulnar side. It was the look of these hands which first suggested to me the idea that the patient might be suffering from Paralysis agitans.

Another symptom is the rigidity. A patient of Charcot's said that his joints appeared "soldered together." Benedikt suggests that the habitual rigidity of a certain number of the muscles undoubtedly contributes in rendering movement laborious.

The muscular rigidity which occurs in Paralysis agitans seems to me peculiar. It is not apparently a spastic condition like that e.g. which we see in the late rigidity of hemiplegia or in the sequel of myelitis with secondary degeneration of the lateral columns of the cord. It resembles to a remarkable extent the stiffness which results from disuse. You see this condition well marked in the muscles of a forearm which has been for some weeks confined to a splint on account of fracture. The muscles are feeble and stiff, but the stiffness can be overcome with tolerable ease by passive movements. There is no tension of the muscles in this case any more than there is in the case of the fractured arm, and here, as you see, they are actually flabby. Along with his rigidity, if such it can be called, is a condition which Charcot has called attention to —a retardation in the execution of movements. The patient performs movements with extreme slowness. "In relation to the faculty of speech," Charcot writes, "there is a comparatively considerable lapse of time between the thought and the act.

One might suppose that the nervous influx cannot be set to work until after extraordinary efforts." Probably, also, I would suggest, the faculty of receiving impressions is likewise defective.

The muscular stiffness is commonly most marked in the advanced stages of the disease. Charcot writes: "There are cases, though these are rare indeed, in which muscular rigidity is a symptom of the early stage of the disease, and a really prominent one. I have recently observed an example which belongs to this category. The patient had scarcely noticed the tremor, which, in fact, showed little intensity in his case, and was confined to one hand. He already displayed, however, in a high degree, the peculiar attitude of the body and its members, the difficulty of movement and the characteristic gait." In a case under the care of Dr. Gowers in this hospital, the patient, a female, exhibited all the symptoms of Paralysis agitans except the trembling, which was barely perceptible. In this man there is an entire absence of tremor. But we do not know whether the symptom has always been absent. It is quite possible that there may have been a certain amount, perhaps too small to attract the patient's attention. However that may be, I do not consider that the total absence of trembling is any bar to our reception of this case as one of Paralysis agitans. The remaining symptoms appear to me to be quite sufficient to enable us to relegate the case to that category. I may remind you of the analogy in this respect with what obtains in another disease of the cerebro-spinal nervous system—tabes dorsalis. The cases are numerous in which the symptom ataxy of gait which gives the name progressive locomotor ataxy to the disease, is entirely absent.

It is very probable that such cases as this man's are not so rare as might be thought. Looking back to past experience, I am disposed to think that I have seen several such in workhouses and hospitals, where they have been classed with cases of softening of the brain.

But the converse to this total absence of tremulousness may be observed. The movements may be so intensified as to occupy almost the whole attention. I lately saw the following case:

Mrs. M., 64, a farmer's widow (April 26, 1881). Twenty-seven years ago she began to get weak in the right hand, and thirteen years since her head began to shake.

Now as she sits she is constantly moving all over; her head is jerked, the arms, legs, and body writhing. She is constantly gasping in her breath, her face twitching—in an incessant state of distress. The larynx is prominent, owing to great action of the sterno-mastoids and trapezii. There is, she says, a "distressing feeling in her inside."

She is constantly obliged to be occupying herself. Whilst I examined her she was knitting.

Her daughter says she is better in herself than she was six years ago, but the movements are about the same.

She sleeps about four hours at night.

The general health is good.

She is "never ill." She always feels better when by herself. Any nervous excitement increases her distress.

There is constant snorting and sniffing.

Here is an example of another variety:

William W., æt. 64, was admitted into the hospital on May 20, 1878, with Paralysis agitans, which had begun five years previously.

He was an engine-driver who had never had a day's illness in his life. In the course of his work he had to crawl into very hot boilers to clean them out, and to this he attributed his illness.

His symptoms began with subjective coldness of the knees, shortly followed by shaking of the left hand, left leg, and then of the right hand.

There was almost constant shaking of the left hand. If he took hold of an object and held it firmly, the shaking stopped for a few seconds, but then recommenced worse than ever.

In this man there was a very peculiar condition. Whilst he was at rest there was no shaking of his legs, but if he stood up and attempted to walk, he remained for a time unable to start, his feet, however, beating the ground rapidly. All of a sudden this would cease, and he would start off at a fair pace, though he required some one to hold him up. This "marking time'

action when he tried to walk had been observed about eight months. The patient, who was an engine-driver, appeared much struck with Mr. Broster's suggestion, that it reminded one of the wheels of a locomotive failing to bite the rails when they are slippery with frost, and making, in consequence, ineffective revolutions.

Paralysis agitans is sometimes acute in its progress, and this, I think, is more often the case when the patient is comparatively young. I lately saw a man, æt. 38, whose symptoms dated from one year only. They had commenced with hesitation of speech, which was followed by shaking, first of the left and then of the right arm, and when he came to me he presented all the symptoms in a marked degree.

There was a patient in the hospital, a year or two ago, who was still younger. I gather the following account from Mr. Broster's notes, taken on his admission:

Benjamin L., æt. 22, was admitted into hospital on April 30, 1879. His occupation is that of a butcher, and for five years past he has been in London, having previously lived in the country. He looks about his age. Somewhat apathetic in manner, he yet complained of irritability of temper, especially when exposed to noise. His memory is good. He has no headache nor giddiness. The speech is thick, and somewhat indistinct; the articulation being hurried, and the words run together. The tongue showed marked tremor. Saliva dribbled from his mouth at times. There is difficulty in swallowing, and he is obliged to have his food chopped up fine.

His face wears a fixed look. He cannot frown, and can only show his teeth feebly by voluntary movement. During involuntary movement, on the other hand, as when he laughs, he shows them very well. He has no emotional instability. As he sits, there is marked shaking of the head and upper limbs. The arms—especially the right arm—are in a continual state of rather violent agitation, which is increased by excitement, and is least when he is left quietly to himself. There are also involuntary uniform movements of the legs and feet when he is seated. If he lifts his heel off the ground, so as to rest solely on the ball of the foot, there is a rapid movement similar to foot-clonus. No foot-clonus can be elicited

by sudden passive dorsal flexion of the foot. The patellar tendon-reflex is normal—free from exaggeration. In his walk he inclines forward, and "scuffles" along, but not so hurriedly as is often seen in these cases. He complains of stiffness in his arms and legs.

When he picks up anything with either hand there is a momentary lull in the movement, and he takes hold of them quietly and steadily. Immediately afterwards the movements recommence. He is very easily pushed forwards or backwards, however much he may try to resist this.

His breathing is of a half-sobbing character.

There is slight lateral curvature. No spinal tenderness.

Now, in this case, it was only eleven months before these notes were taken that the patient had first noticed a slight shaking of the right hand. This very gradually increased, and in three months he was forced to give up his occupation. Two months later he noticed that his left hand was beginning to shake like the right, and after two or three months more his legs also became involved. There was shaking whilst he sat, and he says that they shook all the more if he bore his weight upon his toes. A month or two after this, his swallowing became affected, and his mouth, according to his account, was always full of saliva. About the same time his articulation became embarrassed.

This man acknowledged a course of life of the most dissipated character. He had lived freely and drunk heavily. From the age of 16 to 21 he had masturbated two or three times a day; and from 21 to 22, about the time of the commencement of his symptoms, had indulged to the greatest excess in sexual intercourse. He had never had syphilis, but had once suffered from gonorrhœa. The family history was a healthy one.

It is worth noting that, in the other case—patient aged 38—there was a history of excessive sexual intercourse immediately preceding the commencement of the symptoms.

The patient, William V., who is now attending the hospital, is affected with shaking palsy, which commenced in his left arm. By the dynamometer, the grasp of the right hand measures 50, that of the left 40.

The movements in this case are of a somewhat unusual

character. They are much larger in extent than is commonly seen, so that as the patient sits he flaps his knees with both hands noisily and roughly. His legs also are affected with coarse movements. His walk is characteristic, the body being bent forward, and the head maintaining a "set" position.

If we ask him to pick up a pen off the table, his arm is still whilst he is stretching it out to grasp the object; but when the hand closes upon it the movements begin to recur with great energy.

Another of our out-patients, a female, presents symptoms which are at first strongly suggestive of this disease, but I do not think she is an example of it.

Elizabeth B. is a widow, 66 years of age, who was strong and well, and had nothing to complain of till ten weeks ago, when she woke up one morning retching and vomiting, and found that she had lost power to a certain extent in the left arm and leg. She found that she could not keep her arm still, and her leg also was constantly shaking. A very aged neighbour who came to her, said, "Don't touch me, or you'll pull me down," the shaking was so violent. She knocked the left knee against the right so violently that it was quite bruised. She remained in bed three weeks, during which the movements were very violent, but afterwards they became less so.

Now, we observe that as she rests her hand on her knee it is steady, but when she tries to pick up things from the table there are rhythmical movements—flexion and extension movements of the hand numbering about 180 in the minute. The attitude of the hand is not that of Paralysis agitans. A friend who comes with her says that she is sometimes obliged to throw the bed-clothes off, she complains so much of heat. Since she has been here before us, her face has come over in a red flush several times.

The wrist-reflex is exaggerated on both sides, but especially on the left, whilst the patellar tendon-reflex approaches to a clonus on the left, and is in some excess also on the right side. She walks with short toddling steps, and is compelled to use a stick, otherwise she falls. The left knee never shakes now, but

sometimes in walking it "catches up." The dynamometer shows a grasp of 40° with the right, 22° with the left hand.

She has never had any numbness in the left hand, but there is often aching pain in the forearm, especially towards night.

We do now and then observe a state of tremor in the paralysed limbs of a hemiplegic patient; but so far as I have seen, it is very rare to find such an amount of tremulous movement as we see in this woman. Here the tremors closely resemble those of Paralysis agitans, and the case might easily be mistaken for one of that disease.

According to Nothnagel, the condition has been observed in connection with lesion in the following localities:—1. The internal capsule in its posterior segment. 2. Optic thalamus. 3. The foot of the corona radiata. In these situations it is, he points out, that lesions are most apt to be accompanied by hemichorea and athetosis, with which it is supposed these tremors are nearly allied. It is not certain whether the condition occurs as a result of cortical lesions.

This patient evidently had a sudden cerebral attack, accompanied by vomiting and loss of power, in the arm and leg of the left side. In the suddenness of its origin, the disease contrasts as strongly as possible with shaking palsy.

The exaggeration of reflex amounting to foot-clonus on the affected side, is also opposed to the diagnosis of that disorder. The attack has, probably, been of an apoplectic character, the exact situation of which, however, I am not prepared to indicate with any confidence.

Passing over the morbid anatomy of Paralysis agitans, about which nothing conclusive is as yet established, I would refer to one or two points in regard to the symptoms.

If we examine the upper extremity in a case of true shaking palsy, we are struck at once by the circumstance that apparently the small muscles, those concerned in the most delicate movements, are those which suffer most. The attitude of the fingers is that which would be produced by alternate contraction and relaxation of the fibres of the interosseous muscles. It is unusual in other conditions to find predominance of affection of such muscles over the larger ones. In the climax of an epileptic seizure the hand is clenched.

The contraction of the large and powerful flexor muscles lying on the anterior surface of the forearm, overpowers completely that of the small interossei. So also in the converse of convulsion—in hemiplegia, when the paralysis is complete the fingers lie passively in an attitude of semiflexion. There is only one condition, I think, in which the position of the fingers at all resembles that which they occupy in shaking palsy, and that is the singular disorder called Tetany. It is not easy to see any kind of association between these disorders, but the point is perhaps worth noting.

As regards the piping senile voice, to which I have already referred, I have lately met with an instance in private practice which has afforded me an opportunity of investigation. The patient, William B., aged 57, has the characteristic fixed look and the piping voice. His right hand, ever and anon, but not constantly, is shaken to and fro. The left also is shaken, but much less. He feels very weak in the right arm, and says that he has scarcely any use at all in it.

He does not come over in flushes of heat or perspiration.

Sometimes, he says, he can scarcely talk. This, he thinks, is owing to something in the throat. His wife has noticed the alteration in the voice.

About two years ago he began to feel loss of power in the right arm—that hand would drop occasionally from the wrist so that he could not depend upon holding things. The loss of power has gradually increased, especially of late. Within the last few weeks he has inclined to trip with the right leg, which feels, as it were, shorter than the other.

Lately, for a fortnight past, he has been liable to giddiness, and on one occasion he nearly fell down.

He does not suffer from sickness. He has usually had good health. His appetite is good; he sleeps well; the bowels act.

He has a difficulty in holding his urine, and would wet himself if he were not very quick. He gets up two or three times in the night to pass water—just a little.

Memory is good. He would rather be by himself, and avoids noise and company. There is a fixed look of the face with half a frown upon it, and the head is held stiffly, the eyes usually looking straight in front.

The tendon-reflex at the wrist is equal on both sides, and not abnormal.

At my request Dr. Felix Semon was good enough to examine this patient with the laryngoscope. His report is that there is neither motor nor sensory paralysis nor loss of reflex-sensibility to be detected in either pharynx or larynx, and that both these parts are quite free from traces of previous specific or other disease. It seems probable, therefore, that the high tone, the piping voice, which I have likened to that adopted by an actor who represents advanced age upon the stage, may be dependent upon a functional imperfection of the soft palate, perhaps due to a vibratile condition of its muscular structure.

DEAN SWIFT'S DISEASE.

BY DR. BUCKNILL, F.R.S.

During the past autumn I received a letter from a gentleman engaged in literary work, requesting my opinion on the "mysterious disease" of the great author and wit, whose name distinguishes this paper. My interlocutor particularly wished to know whether the sane part of Swift's life was likely to have been in any way affected by the latent presence of insanity; whether a correct diagnosis was possible; whether parallel cases were on record; and, finally, whether a surfeit of green fruit, at the age of twenty-three years, was capable of resulting in the absolute fatuity from which the patient suffered at seventy-five.

This questioning has stimulated me to an investigation which I had thought was already threadbare, but which I found full of interest; and when I say that, upon weighing the evidence, it will probably be acknowledged that Jonathan Swift's mysterious disease was an instance of that curious form of disease, Labyrinthine Vertigo, or Le Maladie de Ménière, the knowledge of which is one of the most recent triumphs of pathological research directed by physiological experiment, it will scarcely be thought that it was needless to reopen a controversy in which already everything had been said which ought to have been said, and not a little which ought not to have been said.

When Dr. Beddoes suggested that Swift's ailments and his conduct towards women were due to dissolute habits in youth, Sir Walter Scott replied, that "until medical authors can clearly account for and radically cure the diseases of their contemporary patients, they may readily be excused from assigning dishonourable causes for the disorders of the illustrious

dead." But if Dr. Beddoes were unquestionably wrong in making such a suggestion without evidence, Sir Walter was scarcely right in making his retort too general; for if medical opinions respecting the states of mind of persons who have departed this life must be forbidden until medical men can ensure the radical cure of diseases, not only will much valuable evidence respecting the validity of wills be excluded, but the science of pathology itself, depending upon the history of diseases and verified by observations made after death, must be interrupted until an event which seems impossible has taken place.

Whether the causes of disease are or are not dishonourable, and whether the subjects of them are or are not illustrious, has nothing to do with the scientific question; and the often-quoted sneer of Swift's greatest biographer at the medical profession seems, when examined, as silly as general sarcasm usually is. Undeterred by such sarcasm, an eminent medical man did investigate the causes for the disorders of the illustrious dead in a work which he modestly called an Essay, published in 1849, and entitled 'The Closing Years of Dean Swift's Life,' &c., by W. R. Wilde, M.R.I.A., F.R.C.S. This little work, marked by the excellencies of careful research, sound reasoning, moderate opinions and fair conclusions, would have rendered further discussion needless if medical science had stood still since its publication; but the advances made in medical psychology during the last thirty-two years might give us some excuse for reconsideration even if Ménière had never made his discovery of a definite form of disease previously unrecognised, which appears to conform in all important points with the lifelong disease of the illustrious Dean.

Even in Swift's latest biography, or rather that fragment of one which so strongly makes us feel that touch of the vanished hand of the most appreciative critic of men of letters, the late John Forster's charming volume, the author speaks of Swift's giddiness and deafness, not as symptoms of one disorder, but as "two lifelong enemies," and it is a curious enough fact that Swift himself attributed the origin of these two enemies to different causes, operating at different periods. In a letter to

Mrs. Howard in 1727 he writes: "About two hours before you were born I got my *giddiness* by eating a hundred golden pippins at a time at Richmond; and when you were four years and a quarter old, bating two days, having made a fine seat further in Surrey, where I used to read, there I got my *deafness*, and these two friends, one or other, have visited me one or other every year since; and, being old acquaintances, have now thought fit to come together." Mrs. Howard having been born in 1690, the date of the deafness given in this letter would be 1694, when Swift was twenty-seven years old. But in a passage quoted by Forster, p. 48, Swift wrote, "In England before I was twenty I got a cold, which gave me a deafness that I could never clear myself of . . . . my left ear has never been well since."—April 30, 1737.

One cannot but concur in Johnson's remark on the above, that "The original of diseases is commonly obscure, and almost every schoolboy eats as much fruit as he can get, without inconvenience." But it may also be remarked that if Swift had been "a contemporary patient," although we might not have effected a radical cure of his disease we should at least have understood enough of its origin and nature to have saved him from tormenting himself by a lifelong abstinence from fruit, of which he was passionately fond, under the belief that it had caused and continued to excite his disease by inducing that "coldness of stomach to which he attributed his vertigo and its accompanying sickness."

In a letter of 1708 he says that, "I was through a long time pursued by a cruel illness that seized me at fits and hindered me from pursuing any business." It is possible that this illness was but a return of the dangerous colic from which he suffered in 1696; and it is not until 1710 to 1713, and while residing in London, that he describes in some detail the symptoms of his lifelong complaint in his 'Journal to Stella.' The most descriptive passage is perhaps the one dated Oct. 31, 1710:—

"This morning, sitting in my bed, I had a fit of *giddiness;* the room turned round for about a minute, and then it went off, leaving me *sickish*, but not very. I saw Dr. Cockburn to-day, and he promises to send me the pills that did me good

last year; and likewise has promised me an oil for my *ears*, that he has been making for that ailment for somebody else."

A fit of giddiness, with sickness and ear-disease, is not this labyrinthine vertigo?

On different days in *Jan.* 1711 he writes:—

"I had an ugly fit in my chamber last night." . . . "My head is not in order, and yet is not absolutely ill; but giddyish, and makes me listless." . . . "One fit shakes me a long time."

Feb. 1.—"I walked into the City to dine, but I walked plaguy carefully, for fear of sliding against my will."

April 18.—"I did not go to the House of Commons about the yarn: my head was not well enough. I know not what is the matter. It has never been thus before; two days together giddy from morning till night, and I totter a little, but can make a shift to walk."

In *May*: "I do not totter as I did, but walk firm as a rock, only once or twice for a minute."

Sept. 1*st* he notes an important peculiarity, distinguishing cerebral from stomachic vertigo:—"My head is pretty well; only a sudden turn at any time makes me feel giddy for a moment, and sometimes it feels very stuffed."

The journals of October show that he distinguished ordinary from vertiginous headache:—"My head has ached a little in the evening, but it is not of the true giddy sort, so I do not much value it." . . . "I had a little turn in my head this morning, which, though it did not last above a minute, yet being of the true sort, has made me as weak as a dog all this day."

During the years of residence in London which embrace the period of the 'Journal to Stella,' his other enemy, deafness, is only referred to incidentally, as when he compares it to that of the Lord Treasurer; but after his return to Ireland, his deafness becomes sufficiently severe to make him complain.

In 1720 he writes:—"What if I should add that once in five or six weeks I am deaf for three or four days."

In 1724 he writes:—"I have been this month past so pestered with a return of the *noise and deafness* in my ears that I had not the spirit to perform the common offices of life." Subse-

quently, in the same year: "My deafness has left me above three weeks, and therefore I expect a visit from it soon." It was evidently periodic and paroxysmal, like the giddiness.

He complains in another letter of an old vexatious disorder of a deafness and *noise* in the ears. In 1827, in a letter to Sheridan, he says that his deafness is worse than it ever before had been, and that it is accompanied by giddiness and tottering. "I believe," says he, "that this giddiness is the disorder which will at last get the better of me." And again, "I walk like a drunken man, and am deafer than ever you knew me."

In 1728, in "about eight months," says Wilde, "he had half-a-dozen attacks of the giddiness and sickness, each of which lasted about three weeks." But in 1731 he wrote to Mr. Gay, "The giddiness I was subject to, instead of coming seldom and violent, now constantly attends me more or less, though in a more peaceable manner, yet such as will not qualify me to live among the young and healthy." In 1736, writing to Pope, "years and infirmities have quite broke me. I mean that *continual* disorder in my head." In 1737, to Alderman Barker, "I am forced to tell you my health is much decayed; my deafness and giddiness more frequent; spirits I have none left; my memory is almost gone."

Long before, however, these symptoms had commenced. Impairment of memory he complained of as early as 1713, after the attack of shingles; and later on in the same year he speaks of his horrible melancholy changing into dulness, and from thenceforth increasing irritability of temper and mental depression are traceable throughout his history and correspondence. Not that he was at any time really of unsound mind or incapable; for when in 1737, in the Bettesworth affair, a gratifying address was presented to him, it is recorded that "when this paper was delivered Swift was in bed, giddy and deaf, having been some time before seized with one of his fits; but he dictated an answer in which there is all the dignity of habitual pre-eminence and all the resignation of humble piety."

The above quotations are but a selection from a far greater number of references which might be made to Swift's letters

and journals, affording conclusive evidence, as I venture to think, that he suffered from twenty years of age from the disease, whose characteristic symptoms are, "that the patient is suddenly seized with vertigo and a feeling of nausea or positive sickness, with great constitutional depression and faintness. Usually the giddiness comes on simultaneously with ringing or buzzing in one or, it may be, both ears."—*Ferrier.*

It has this year been pointed out by Féré, in the 'Revue de Médecine,' that there are two forms of the disease to be recognised, "une forme grave avec état vertigineux à peu près permanent interrompu par les paroxysmes, et une forme moins fâcheuse, constituée par des accès séparés par des périodes de santé parfaite." "Dans la forme bénigne [of which Swift's was an example] les accès ne se produisent quelquefois qu'à des distances très éloignées. E. Ménière cite une malade qui eut une rémission de onze mois. Pendant ces périodes d'accalmie, la surdité persiste avec une intensité variable, et elle s'accompagne souvent des sensations subjectives intermittentes de l'ouïe. La maladie elle-même dure tant que la surdité n'est pas absolue."

Up to the date to which we have traced the progress of the disease, it appears to have been purely a physical malady, with no mental symptoms, unless some degree of loss of memory can be so called. Swift, indeed, complains bitterly of the impairment, as if memory were gone, and in his declining years of age and sickness it may have been a dull function compared with the brilliant faculty he once possessed. But clearly the memory was still serviceable which enabled him to compose, with wonderful vivacity, even such poetry as that outburst against political and social corruption—'The Legion Club'—which Jeffrey thinks "deserving of attention as the most thoroughly animated, fierce and energetic of all Swift's metrical compositions; and though the animation be altogether of a ferocious character, and seems occasionally to verge upon absolute insanity, there is still a force and a terror about it which redeems it from ridicule, and makes us shudder at the sort of demoniacal inspiration with which the malison is vented." This poem, written in 1736, was his last work; its appreciation by his most hostile critic will show how little he suffered from loss of any mental faculty when he wrote it.

That disease and grief had made him irritable and passionate, and often desponding, is clear enough from his correspondence and the accounts of him which have come down to us; but that there was any failure of mind this 'Legion Club' fully disproves; and if fiercely expressed hatred is any evidence that an author is on the verge of insanity, Jeffrey must have been curiously insensible to the testimony he was bearing against his own soundness of mind in his criticism of the greater master of his own art.

Between 1736, when Swift wrote 'The Legion Club,' and 1741, when the outbreak of insanity really took place, there can be no doubt that he passed through a period of great wretchedness and depression—he was "miserably ill." He had lost to a great extent two of his senses, for he was deaf and his eyesight failed; his dearest friends had died before him, and his public sympathies were constantly outraged.

In 1738 he wrote to Alderman Barker, "I have for almost three years past been only the shadow of my former self, with years of sickness and rage against all public proceedings, especially in this miserably oppressed country. I have entirely lost my memory, except when it is aroused by perpetual subjects of vexation."

Two years later he wrote the following pathetic letter to his old friend Mrs. Whiteway:—

"I have been very miserable all night, and to-day extremely deaf and full of pain. I am so stupid and confounded that I cannot express the mortification I am under, both of body and mind. All I can say is that I am not in torture, but I daily and hourly expect it. Pray let me know how your health is, and your family. I hardly understand one word I write. I am sure my days will be very few; few and miserable they must be. I am for these few days yours entirely,

"J. Swift."

"If I do not blunder it is Saturday."

A very pitiful state this period of becoming insane, and yet not having become so. But even at this late date one cannot recognise the invasion of mental disease. Misery and despondency there was, more than enough, but not madness, unless Job was mad. But Swift was rapidly tending towards

madness, and he knew it, for strong forebodings of insanity, which are not common, existed in him in a remarkable degree. Sir Walter Scott says that "his first state was that of violent and furious lunacy;" but Sir William Wilde points out that all the biographers have had no other sources of information as to the outbreak and history of his insanity than two letters; one from Mrs. Whiteway, and one from his cousin, Mr. Deane Swift.

Mrs. Whiteway wrote Nov. 22, 1742, as follows, omitting some expressions of feeling:—

"I was the last person whom he knew, and when that part of his memory failed he was so outrageous at seeing anybody that I was forced to leave him, nor could he rest for a night or two after seeing any person. He walked ten hours a day, would not eat or drink if his servant stayed in the room. His meal was served ready cut, and sometimes it would be an hour on the table before he would touch it, and then eat it walking. About six weeks ago, in one night's time his left eye swelled as large as an egg, and the lid, Mr. Nicholls [his surgeon] thought would mortify, and many large boils appeared on his arms and body. The torture he was in is not to be described. Five persons could scarce hold him, for a week, from tearing out his own eyes; and for near a month he did not sleep two hours in twenty-four. Yet a moderate appetite continued; and, what is more to be wondered at, the last day of his illness he knew me perfectly well, took me by the hand, called me by my name, and showed the same pleasure as usual in seeing me. I asked him if he would give me a dinner? He said, 'To be sure, my old friend.' Thus he continued that day, and knew the doctor and surgeon and all his family, so well that Mr. Nicholls thought it possible he might return to a share of understanding, so as to be able to call for what he wanted, and to bear some of his old friends to amuse him. But alas! this pleasure to me was but of short duration; for the next day or two it was all over, and proved to be only pain that had roused him. He is now free from torture, his eye almost well, very quiet, and begins to sleep, but cannot without great difficulty be prevailed upon to walk a turn about his room; and yet in this way, the physicians think, he may hold out for some time."

The only other authentic account from personal knowledge is contained in the letter of Mr. Deane Swift to Lord Orrery, dated April 4th, 1744. After stating that a thousand stories of the illness had been invented and imposed upon the world, he proceeds to state some facts witnessed by himself.

"On Sunday the 17th of March, as he sat in his chair, upon the housekeeper removing a knife from him as he was going to catch at it, he shrugged his shoulders, and rocking himself, he said, 'I am what I am. I am what I am;' and about six minutes afterwards repeated the same words two or three times over. Sometimes he will not utter a syllable, at other times he will speak incoherent words; but he never yet, as far as I could hear, talked nonsense, or said a foolish thing. About four months ago he gave me great trouble. He seemed to have a mind to talk to me. In order to try what he would say, I told him I came to dine with him, and immediately his housekeeper, Mrs. Ridgeway, said, 'Won't you give Mr. Swift a glass of wine, sir?' he shrugged his shoulders, just as he used to do when he had a mind that a friend should not spend the evening with him. Shrugging his shoulders, your Lordship may remember, was as much as to say, 'you'll ruin me in wine.' I own I was scarce able to bear the sight. Soon after he again endeavoured, with a good deal of pain, to find words to speak to me; at last, not being able, after many efforts, he gave a heavy sigh, and I think was afterwards silent. This puts me in mind of what he said about five days ago. He endeavoured several times to speak to his servant [now and then he calls him by his name]; at last, not finding words to express what he would be at, he said, 'I am a fool.' Not long ago the servant took up his watch that lay upon the table, to see what o'clock it was; he said 'bring it here,' and when it was brought, he looked very attentively at it. Some time ago the servant was breaking a large stubborn coal, he said; 'that's a stone, you blockhead.' In a few days, or some very short time after guardians had been appointed for him, I went into his dining-room, where he was walking; I said something to him very insiguificant, I know not what, but, instead of making any kind of answer to it, he said, 'Go, go,' pointing with his hand to the door, and immediately afterwards, raising his hand

to his head, he said, 'My best understanding,' and so broke off abruptly, and walked away."

These two letters are stated by Sir William Wilde to be the only account of the last three years of Swift's life that has come down to us. He died October 19th, 1745, in the seventy-eighth year of his age. His death, according to Lord Orrery, being easy, without the least pang or convulsion; but according to Faulkner, being one of "great agony, with strong convulsive fits" for thirty-six hours before. The only record of the autopsy which was made is that Mr. Whiteway "opened the skull, and found much water in the brain." A more interesting record, however, remains in the plaster cast of Swift's head. Of this Sir Walter Scott says that "the expression is most unequivocally maniacal, and one side of the mouth horribly contorted downwards as if in pain." But Sir William Wilde, whose observation we greatly prefer in such a matter, says, "The expression is remarkably placid, but there is an evident drag on the left side of the mouth, exhibiting a paralysis of the facial muscles to the right side. Upon the back of the cast are two lines of writing, greatly defaced, of which this much can still be read, 'Dean Swift, taken off his * * * * the night of his burial, and the f * * * * one side larger than the other in nature * * Opened before * * * The mould is in pieces.'" A deep indentation, says Wilde, shows where the calvarium had been sawn; and accurately corresponds with the division of the skull found in Swift's coffin in 1835. On the same excellent authority we know that the cast of the interior of Swift's skull is remarkable as showing the enormous development of the vessels within the cranium, the very small anterior lobes, the great size of the glandulæ pacchioniæ, the exceedingly small cerebellum, a natural formation as may be seen by the very low position of the tentorium, the immense size of the posterior and middle lobes, particularly the former, and the absence of any appearance of disease in the anterior lobes, as far at least as this cast of the interior of the skull is capable of demonstrating.

Swift of course was not exempt from intercurrent diseases, the most important of which appear to have been a severe colic in 1696, which brought him to extremity, "so that all

despaired of my life, and the newspapers reported me dead;" an attack of herpes zoster of the left neck and shoulder, with its atrocious after-pains, in 1712; and a severe attack of ague in 1720, which lasted a whole year. But these maladies appear to have passed away without permanent injury, and it is to the cold which, before he was twenty, gave him a deafness and an ear "which has never been well since," to which we must look as the origin of his physical and ultimately of his mental miseries, and which made him often describe himself as

"Vertiginosus, inops, surdus, male gratus amicis."

He does not appear ever to have guessed that his vertigo was due to the state of the auditory organ, nor indeed did any physician ever suspect that such was the case in any one suffering from ear-giddiness, until, led to reflect on the subject by Fleuren's experiments on the semicircular canals of pigeons and rabbits, Ménière recognised the causal connection in 1861. The paroxysmal nature of the affection, with long intervals of immunity, is well marked in Swift's case, although it would appear from a quotation made by Forster, p. 253, from one of his note-books, that the fits were pretty frequent, the reference to fits in his journal and correspondence only applying to the more serious occasions. The quotation is as follows:

"1708. *Nov.*—From 6th to 16, often giddy. G^{d} help me. So to 25 less. 16, Brandy for giddiness 2*s*. Br^{dy} 3^{d}. *Dec.* 5.—Horribly sick. 12*th*.—Much better, thank God and M. D.'s prayers. 16*th*.—Bad fit at Mrs. Bartons. 24. Better; but dread a fit. Better still to the end. 1709, *Jan.* 21*st*.—An ill fit but not to excess. 29. Out of order. 31. Not well at times. *Feb.* 7.—Small fit abroad. Pretty well to the end. *March*.—Headache frequent. *April* 2*nd*.—Small giddy fit and swimming in the head. M. D. and God help me. *August*.—Sick with giddiness much. 1710. *Jan*.—Giddy. *March*.—Sadly for a day. 4*th*. Giddy from 4th. 14*th*.—Very ill. *July*.—Terrible fit. G^{d} knows what may be the event. Better towards the end."

It is true that the paroxysms were not so numerous as those of most other cases of the disease which are on record, and also that the deafness never became absolute, and therefore

that the disease never ceased. It increased in intensity as life advanced, until it confined him to his chamber for weeks at a time. It is also to be remarked that a slight degree of vertigo caused great constitutional disturbance. "I had a little turn in my head this morning, which, though it did not last above a moment, yet, being of the true sort, has made me as weak as a dog all this day."—*Journal*, Oct. 23, 1711. "This morning I felt a little twitch of giddiness, which has disordered and weakened me with its ugly remains all this day."—*Journal*, Jan. 25, 1812. Another characteristic of the vertigo, noted in a quotation already given, is that at one time any slight movement of the head brought it on.

It is certain that this fearful disease, aggravated with the increase of years, had an influence in the causation of Swift's insanity; but that its influence was direct, that is to say by the extension of the local disease to the brain, is by no means so sure as its indirect effect as one source of the profound depression which marked the latter years of his sane life. We have no authentic account of the first outbreak of insanity, and Sir Walter's statement that, after his understanding failed, "his first state was that of violent and furious lunacy," would seem to have been applicable only to that later period when he suffered indescribable torture from some unknown local disease, producing exophthalmos of the left eye. It is clear there was emotional depression amounting to melancholia, and "much water in the brain," which was probably sub-acrachnoid effusion, is sufficient evidence of dementia. But there was also that form of aphasia in which scraps of reasonable language come automatically, though intentional effort can produce no words, and very curiously in connection with this fact comes the evidence of the plaster-cast, "brought to light a hundred years after death," that there was right-sided hemiplegia. The knowledge of the importance of this fact also has been acquired since Sir William Wilde wrote his work, and it is not therefore surprising that while he so carefully and skilfully marshals the data upon which our diagnosis is now made, he does not connect the right-sided hemiplegia with the very peculiar affection of speech recorded by one of the two authentic witnesses above quoted. Sir William Wilde ex-

presses the opinion that the hemiplegia had existed for several years before death, "for we find the same appearance much glossed over by the artist, together with a greater fullness or plumpness of the right cheek shown in a very admirable bust, probably the last ever taken." But as Wilde admits, the "six well-known busts of Swift, undoubtedly taken about the same time, exhibit six different forms of head bearing but little resemblance to each other," the much glossed over appearance can therefore scarcely be admitted as evidence. Probably the stroke of palsy recorded in the plaster cast had taken place unobserved at or about the time of the actual outbreak of the mental disorder, which might have masked the physical symptoms from observation.

When "the Vandal desecration of monuments" in 1835 exposed Swift's skull to the phrenologists, the great Dublin aurist might possibly have found in the bones of the ear traces of the cause of his giddiness. When Mr. Whiteway examined the brain, he might have found the cause of Swift's right-sided hemiplegia and his aphasia. It is enough now that we can diagnose his lifelong disease as labyrinthine vertigo, and his insanity as dementia with aphasia; the dementia arising from general decay of the brain from age and disease, the paralysis and aphasia from disease of one particular part of the brain.

With all the tortures of the lifelong disease from which he suffered, and its obvious effect upon his temper in his later years, it is wonderful that Swift did retain his reason until, in the seventy-fourth year of his age, he was in all probability struck down by a new disease in the form of a localised left-side apoplexy or cerebral softening, which determined the symptoms of his insanity.

That Swift's works contain no indications of insanity appears to me certain. As well say that Shakespeare was mad because he wrote a good deal which we think nasty. In the fashion of the day, Swift was too prone to make what may be called excrementitious jokes and jibes. But that perfect gentleman Antonio voided his rheum upon Shylock's beard; and the same kind of thing runs through our literature, no one objecting, until we rather recently began to become less natural and more

nice. Some of our smaller humourists and men of letters have criticised this great king of humour as if he were both bad and mad, not perceiving that if he were really insane he must be pitied and not cursed. But it is the weakest of arguments to say, with Festus, for want of argument, "much learning doth make thee mad." There is always weakness in madness, but there is little sign of this in Swift's works. There is always some inconsequentness or incoherency in madness, but there is none of this in Swift. Down to that last letter to Mrs. Whiteway he is most wretched, but he is still collected and wholly himself.

One final consideration is that the oppressive and disabling nature of Swift's lifelong disease has been greatly underrated in the more severe of the criticisms which have been made with regard to his conduct to Esther Johnstone. I do not know that labyrinthine vertigo would necessarily incapacitate a man for the performance of marital duties, but it certainly might be a barrier to them more formidable than unprofessional critics are likely to suppose possible. Dr. Beddoes suggested that Swift was impotent from youthful dissipation, of which there is not a tittle of evidence. May not the great and grave disease of which I have adduced such copious evidence have been the real reason why Swift did not live with the woman whom it is certain that he loved with the most tender and persistent devotion?

Critical Digests and Notices of Books.

Etudes Cliniques sur l'Hystéro-épilepsie, ou grande hystérie. Par le Dr. PAUL RICHER. Paris, Delahaye, 1881: xvi.-734 pp.

THE activity displayed in the study of hystero-epilepsy and the curious phenomena allied to it, has resulted in the formation of a large literature, in the shape of innumerable papers and articles dispersed in numerous periodicals. The interest taken in the subject goes on increasing every day, and Dr. Richer deserves our best thanks for having undertaken the task of reducing the multitude of loose material to an orderly though comprehensive digest. Our aim here will be less to criticise than give a condensed account of the work before us.

The typical hystero-epileptic fit is usually heralded by a period of prodromal symptoms, affecting one or more functions. Thus flatulence, spasm of the throat, ptyalism, dyspnœa, palpitation, vasomotor disturbances, and other similar signs may be present in the sphere of organic life. The muscular system may be the seat of various symptoms, such as loss of power, tremors, transitory or persistent contractions. In the sensory sphere, anæsthesia, complete or partial (frequently affecting one-half of the body), and involving the special senses as well as the common cutaneous sensibility. Painful symptoms are not rare, constituting the aura. The latter, as Charcot has shown, stands in intimate connection with the ovarian tenderness, which is pretty uniformly present at all times in hystero-epileptics. Pressure upon the tender spots calls forth a series of very peculiar sensations, which follow a definite course, clustering at first about the ovary, next about the throat, and finally in the brain, and are the same as those observed in the spontaneous aura. Many patients present other points on the

body besides the ovarian region, pressure on which is followed by the aura and the fit. These have an obvious resemblance to the epileptogenous zones of Brown-Séquard's guinea-pigs, and hence have been called hysterogenous zones. These zones are very variously distributed, and not nearly so frequently confined to the hemianæsthetic side as the ovarian tenderness is. It may be added here, that pressure on all these points are frequently as effectual in cutting short the fit—by substitution or interference, as it were—as in determining it. Lastly, among the prodromal phenomena, we must mention psychical disturbances. The moral and intellectual processes are variously distorted, and hallucinations play an important part in the picture. These take sometimes an erotic turn of remarkable violence, leaving the patient during her conscious hours under a vivid impression of their reality; but more frequently consist in visions of animals and the like, which, curiously enough, persistently make their appearance on the hemianæsthetic side.

It has been the merit of Prof. Charcot to seize, among the apparently incoherent, proteiform phenomena of the hystero-epileptic attack, upon a fundamental order of sequence, to which they can usually be reduced. His generalisations have been amply confirmed by the subsequent observation of new cases either at the Salpêtrière itself, or in other hospitals both in France and abroad, as well as by the careful study of records made by competent observers of earlier times. The records of the demoniac possessions which played such an important *rôle* during the Middle Ages, yield many unexpected points of resemblance between the phenomena occurring under so very dissimilar circumstances, and described in so very different a spirit, and those which form the subject-matter of the volume before us.

The first period of the hystero-epileptic fit is the only one which bears any resemblance to the true epileptic seizure; but, it must be confessed, this resemblance is very close indeed. The prodromal phenomena culminate into loss of consciousness, arrest of respiration, and muscular rigidity. We cannot enter here into any details of the phenomena observed, but must refer the reader once for all to the beautiful illustrations

and the myographic tracings with which Dr. Richer has enriched his book with no stinted hand. Let it suffice for us to say here that the epileptoid period consists of several phases characterised—(1) by tonic contractions with large, slow, partial movements, and presently by universal tetanic immobilisation; (2) by chronic contractions; (3) by resolution. All this, as we said, is very like true epilepsy, but is differentiated from it by the absence of the peculiar cry often uttered by the patient in the latter disease at the beginning of the fit, and by the possibility of bringing the patient to consciousness by ovarian pressure, or voltaic alternatives of a galvanic current.

The second period of the fit succeeds closely to the first, and is aptly designated by Charcot under the name of Clownism. The patient is no longer unconscious, as proved by the fact that if brought round by ovarian compression she is able to recollect what was passing through her mind—though still insensible to the usual external stimuli; she passes through a phase of contortions and a phase of *grands mouvements.* During the first, a tonic spasm of considerable duration throws the body into a series of attitudes defined as "illogical" by Charcot (from their non-correspondence to any emotional or intellectual states). A common form of these attitudes is the *arc de cercle,* or one of its derivatives. When fully developed, the latter consists in a complete opisthotonos, the patient resting upon her toes and forehead. During the phase of the *grands mouvements* there occurs, after a piercing cry has been uttered once or oftener, first a series of more or less rhythmical movements of great amplitude (the legs and trunk, for instance, being thrown high up in the air, or the head and trunk "*saluting*"), and next a scene of the utmost fury, during which the patient yells like a wild beast and struggles wildly, tearing up everything, striking out, and twisting her body in all directions. This part of the attack is the one which may assume the characters of a true demoniacal possession.

We now come to the third period, that of the *passionelles* or emotional attitudes. Here the patient's mind is the prey of hallucinations, which she acts outwardly by throwing herself into the appropriate postures. She is completely insensible

to external stimuli, but when brought out of her fit can still remember what was passing through her mind at the time, and give an account of it which fully tallies with the external manifestations observed. During this period there is frequently a copious flow of words, which give a clue to the different scenes through which her hallucinations carry her. A great proportion of these usually take their origin in memories of her past existence. Events she has witnessed, persons she has known are mixed up with visions of purely imaginary objects, and every passion of the human mind finds its delineation in the remarkable display of "poses plastiques" through which the patients goes during her delirium. Religious ecstasy, joy, anger, spite, sorrow, love in all its phases, from the purest affection down to the most cynical lubricity, with a hundred transitional forms, and every possible combination, all are embodied with the art of the most consummate actor. The flow of thought is often remarkably rapid, and the passage from one mood into its opposite sometimes sudden in the extreme.

The transition between the period of emotional attitudes and that of waking consciousness is bridged over by the fourth period, that of delirium. Here the patient acts less and talks more. Usually the memories of her past life furnish the themes of long soliloquies; but the fact that the consciousness of external things is returning is proved by the illusions to which the patient becomes subject: sights and sounds are perceived, but perverted into factors of her morbid train of thought. Hence the delirium of the fourth period is far more diversified than that of the third, which in many cases is stereotyped, so to speak, the same events here ever recurring in the same order in the mind of the patient, and giving rise to the same display of embodiments of psychical states.

The delirium of the last period, however, is not always uniform and quiet; occasionally hallucinations of variable nature trouble the patient. The visions are sometimes placid, sometimes horrible; the latter are chiefly of animals (zoöpsia). Another feature which often marks the end of the fit is the occurrences of generalised or partial muscular spasms. The patient's body may be twisted into every conceivable shape.

But the following differences may be noted between these and the truly epileptoid contractions; first, that the muscles of respiration are here unaffected; second, that consciousness is present, and the patient is suffering intense pain. On the other hand, old standing hysterical contractures (and paralysis) have been known to disappear at this stage of the fit, which, we may add, may be protracted over some hours or even days.

The typical hystero-epileptic fit, as just described, with its four periods distinctly characterised, is not often met with. It is subject to many variations. For instance, patients often instead of having a single seizure, fall into an *état de mal,* in which numerous fits follow one another, not by mere succession, but by imbrication as it were; that is, the next fit breaks out before the previous one has run through the complete cycle of phenomena. In other cases one (or more) period is developed at the expense of the others, which may be absent, or at least but very transient and faintly accentuated. Others again present phenomena due to the interference of lethargic, somnambulistic, or cataleptic accidents, more or less developed in the course of evolution of the fit.

The first or epileptoid period may predominate, for instance. The patient then may fall into an epileptoid *état de mal,* lasting days or weeks, during which the temperature remains nearly normal (in true epilepsy this condition is marked by a considerable rise). Or she may present a series of incomplete epileptoid fits, similar to a true epileptic vertigo, with a few localised muscular contractions. When the contortions of the second period are carried to their utmost pitch, and accompanied by more than the usual manifestations of the wild and blind fury which we have seen mentioned above, a true "demoniacal possession" is witnessed. Or again, the fearful character implied in this term may be wanting, and mere "clownism," but pushed to its highest degree of development, is observed. The display of muscular power and violence in such cases is perfectly astonishing on the part of otherwise harmless, and it may be delicate, women.

The third period may represent wholly, or nearly so, the hystero-epileptic fit; and then we have in some cases the spectacle of a religious ectasis, in which the patient goes

through the performance of a whole series of acts connected with her religious belief and emotions. The Roman Catholic hagiography is rich in accounts of such cases; and no doubt those of the modern saintly women alleged to be the object of special divine favours, and to prove their claims by exhibitions of this kind, who happened not to be impostors, were simple hystero-epileptics.

Under the name of acute hysterical madness, Griesinger describes the fourth period, that of delirium, when it forms the bulk of the hystero-epileptic fit. It is to be noted that though at times some spasmodic or other factor of the fit accompanies hysterical delirium, the latter may supervene quite independently. Dr. Richer mentions the following points with reference to it:—Conscientiousness of the delirium; analogy with tonic delirium and with somnambulism; influence of present preoccupations and past emotions; exaltation of the intellect; nobility of the ideas; perversion of thought and feeling; fixed ideas; automatism; irresistible impulse; simulation; agitation and stupor. We have spoken above of the frequency of erotic ideas and of hallucinations and illusions.

The readers of 'BRAIN' will find in Vol. I., p. 558, a short account of the first experiments made by Prof. Charcot on hypnotism and the allied conditions, in hystero-epileptics. Dr. Richer gives us a complete summary of all that has been done at the Salpêtrière in this direction. Those who have had the advantage of witnessing the phenomena themselves will agree that they are of the highest promise towards the eventual elucidation of many physical problems, and are an irrefragable proof of the very small part played by "expectant attention" in the once much-abused performances of hystero-epileptics at the Salpêtrière.

The phenomena observed may be grouped under four headings: 1. Cataleptical state; 2. State of suggestion, or automatism; 3. State of lethargy, with muscular hyperexcitability; 4. The same without hyperexcitability, or somnambulism. The author fully illustrates these conditions, and explains the various modes of production and characteristic manifestations of each. We can here give but a succinct account of the facts observed, and commend Dr. Richer's pages to the attention of

the reader interested in the question. Many hysterical patients are thrown, by fixing their eyes upon a bright light, into a state of catalepsy similar to the idiopathic condition so named. There is absolute anæsthesia, waxy flexibility of the limbs, and the curious adaptation of the facial expression to the attitudes given to the limbs. Thus, prayer or threat, love or disgust, may be successively depicted on the countenance when the arms are placed in the corresponding positions. Closure of the eyelids, or sudden removal of the light, converts the cataleptical into a lethargical condition (somnambulism, magnetic sleep, &c.). Here complete muscular resolution prevails, often accompanied with muscular hyperexcitability to the weakest stimuli. Touching or rubbing the skin at any point throws the underlying muscle into a state of contraction, exactly as localised faradisation would. Each of the small muscles of the ear, face, or hand, for instance, may be singled out; or if the facial or ulnar nerve be irritated by pressure, all the muscles innervated are thrown into violent contraction. This contraction may persist for a very considerable time (in the case of the trunk and limbs, not the face) after the patient has been awakened, and presents the same phenomena of transfer as the idiopathic hysterical contractions. During this lethargical condition, clonus of the upper eyelid and of the eyeball-muscles is observed. The anæsthesia is complete. Then follows a condition more allied to somnambulism, in which the patient may be made to perform various acts to order. To awaken the patient it is enough to blow in her face, or press over the ovarian region. The relationship between the lethargical condition and the true convulsive attack is shown by the epileptoid phenomena which accompany its beginning and its end: pharyngeal spasm, foaming at the mouth, &c.

The same patient may be made hemicataleptic and hemilethargic at once by closing one of her eyes when cataleptised in the usual way. The side of the body on which the eye is closed becomes lethargic, whilst the other remains cataleptic.

The contractures produced during the sleep may be resolved by stimulation of the opponent muscles, or by awaking the

patient. They are fixed by bringing her back into the cataleptical condition before awaking her.

Other agents, besides light, cataleptise hysterical patients; thus, the vibrations of a tuning-fork fixed on a resonator, and a sudden loud sound are very effectual. Cases are recorded where patients were seized on hearing the cymbals suddenly used in an orchestra.

Pressure upon the eyeballs, of the fixing of any near object by the patient, produces the lethargic condition, which is readily converted into the cataleptical by merely raising the eyelids; whilst pressure on the vertex gives rise in most hystero-epileptics to a variety of the lethargical condition characterised by the absence of muscular hyperexcitability, and the impossibility of converting it into catalepsy by raising the eyelids. The patient also presents certain psychical phenomena more allied to true somnambulism. The cataleptical condition may be after a while readily made to pass into a state of automatism, in which, though unconscious, the patient may be made to perform series of actions connected with impressions produced on her senses. Thus she can be made to reproduce, with the accuracy of the image in the looking-glass, the movements of the experimenter placed in front of her, or carry out series of movements suggested to her by placing her limbs in appropriate attitudes, such as climbing up a ladder, creeping on all-fours, and the like. Or again, if an object of known use be placed in her hands, she immediately sets herself to carry out the train of actions suggested by it. It is easy also to call forth hallucinations in her mind by verbal suggestions; whispering in her ear, "Here are flowers," "There comes a serpent," and the like, suggests whole trains of thought which give rise to appropriate actions and language, frequently of the most lively description.

It is to be remembered that all patients are not susceptible of being roused out of the cataleptical condition, and that whilst in the lethargic state some may also be made to display the phenomena of automatism. The lethargy without muscular hyperexcitability presents also some curious phenomena upon which the author wisely abstains to dwell, because they are as yet but little explored, and might readily be turned to account

by charlatans. Thus the patient may be made to answer questions, not only with sense, but sometimes far more intelligently than in her waking condition. She performs acts with the greatest accuracy, and is readily thrown into subjection to one individual, who then alone has the power of starting the automatic actions. Nay, each half of the body may be so subjected, independently, to two experimenters.

We cannot enter into any details with reference to the varieties of the hystero-epileptic attack due to the idiopathic immixtion of lethargic, cataleptic, and somnambulistic phenomena ; nor upon the analogies between the hysterical delirium and the cerebral effects of certain drugs, such as hachisch, alcohol, absinthe, opium, &c. The reader will find these topics fully illustrated and discussed in Dr. Richer's work. In the third part he also discusses various topics, which, being probably familiar to our readers (hysterical anæsthesia, contractures, &c.,) we pass over to reach the last chapters of the work concerning the diagnosis and treatment of hystero-epilepsy. Much has been said about the influence of simulation and expectant attention in the causation of the phenomena attending hysterical manifestations. Several writers in England, and elsewhere, who, by the way, did not always take the trouble of going over to Paris to investigate personally the question, have assumed that Prof. Charcot and his disciples were entirely ignorant of the sources of fallacy attending their researches. Dr. Richer's volume will do much to dispel any such impression that may yet subsist. The results of numerous independent tests that have been applied to the phenomena, and the uniformity displayed in the order of their manifestation, are amply sufficient to carry conviction into any unprejudiced mind.

The following points are pathognomonic in distinguishing true epileptic from hystero-epileptic attacks. The latter may be arrested by pressure over the ovarian and other regions of the body, and by electrical shocks (voltaic alternatives), but are not influenced by the bromides. The temperature remains about normal in the hystero-epileptic *état de mal;* and patients suffering from this disorder do not, even after years, present any of the psychical disturbances so common among the true epileptics.

The therapeutics of hystero-epilepsy fall under two heads: 1st, management of the attacks; 2nd, treatment of the patients between the attacks. The former consists in cutting short the fits, or diminishing their frequency, by such measures as compression of the ovaries with the hand or tourniquet, electrical currents, and inhalations of chloroform, nitrate of amyl and the like. The latter, more successful than might be supposed, considering the fearful manifestations of the disease, consists chiefly in the removal of the patient from her often unsatisfactory surroundings and, in addition to moral influences, hydrotherapic and electric applications. The latter are chiefly of the static order, and Dr. Vigouroux (who has charge of this department at the Salpêtrière) has obtained some very interesting results. The static bath (or charging of the patient on an insulated stool) has very marked effects on the hemianæsthesia, for instance, and produces immediately the phenomenon of transfer with consecutive oscillations.

The historical appendix which concludes Dr. Richer's book is by no means the least interesting part of it. He intends to show that hystero-epilepsy is not a new disease, as might be thought from the silence of the earlier medical observers, but that, under various names, forms, and circumstances, it has existed from early times. Striking analogies will be found, for instance, between the descriptions given of the epidemic chorea of the middle ages (14th century) and those of the present hystero-epileptic phenomena. Again, the demoniacal possessions, of which we have witnessed examples within the last few years. The singular epidemic which broke out in Paris 150 years ago, that of the "Convulsionnaires de Saint-Médard," has been carefully described by competent observers, and offers many typical instances of the disease as now observed at the Salpêtrière. In Protestant countries such phenomena are of course unfrequent; still, the scenes witnessed at American "camp meetings," at Irish "revivals"[1] have been so frequently characterised by peculiar sensory and motor disturbances on the part of those who took part in them, that we cannot ignore the neurosis which lay at the root of such manifestations. Again, the ecstatic epidemic of Sweden (1841),

[1] See 'Christian Revivals; their History and Natural History.' By J. Chapman, M.D., M.R.C.P. London, 1880.

and the lives of "holy" women from Douceline (1274) to Louise Lateau (1868), furnish the author with many points of comparison between the so-called religious and pathological phenomena he has so well described.

Dr. Richer is a first-class artist, and his work is made far more instructive, as well as more attractive, by the numerous plates and illustrations which so faithfully reproduce the various phases of the hystero-epileptic fit in all its varieties. The volume before us is an able, complete, and trustworthy account of all that is known of the subject it professes to treat.

A. DE WATTEVILLE.

Kundrat on Porencephalie. (Die Porencephalie eine Anatomische Studie. Von Dr. HANNS KUNDRAT. Graz, 1882. 4to.)

THIS work is a monograph on the morbid condition of the brain, to which the name Porencephalie was first given by Heschl. It is characterised by the occurrence, mostly on the surface of the cerebral hemispheres, of hollows due to loss of substance, either opening into the arachnoid cavity, or separated from this by the visceral arachnoid, extending to various depths into the medullary substance, reaching the ependyma, or even penetrating completely into the ventricle. These defects were for a long time considered of a congenital nature. Yet this is not universally so, nor is their origin thereby explained. To this point our author specially directs his attention. He has been fortunate enough to obtain as many as twelve original cases of his own, and as well as to examine the collection made by his predecessor Heschl. Besides these, reference is made to cases reported by Cruveilhier and others with sufficient precision or illustration as to render them available for his purposes.

In all he deals with forty-one cases.

He finds that porencephalie is an anatomical defect produced by destructive lesions, which in their origin and nature do not differ from those caused by hæmorrhage, thrombosis, embolism or anæmia. This peculiarity consists only in their

seat on the surface, and in their form, which depends on the completeness of the destructive process. These defects may occur *in utero*, and are therefore congenital, or they may be developed at any period of extrauterine life. The former are by far the more frequent and typical. They affect the cerebral cortex, and are conditioned mainly by anæmia of certain cortical arteries, most commonly of the middle cerebral. When hydrocephalus is also present, the affection may extend over the whole cerebral mantle as far as the basal ganglia. When congenital, these defects injuriously affect the development of the brain. They cause changes in the direction of the convolutions, the convolutions disposing themselves radially to the centre of the hollow. They also impair the development of the individual, and lead generally to idiocy, and frequently to inability to use articulate speech.

If the motor regions of the cortex are affected, paralysis with contracture is always produced.

The cases which occur in extra-uterine life, much less common than the preceding, are mostly caused by embolism or hæmorrhage, but may be the result also of injuries. These do not disturb the general form of the brain and the disposition of the convolutions, but when they occur at an early age they prevent normal development and lead to idiocy. In these cases there may be aphasia, but not absolute abolition of the speech faculty. Paralysis with contracture occurs also in these cases when the motor zone of the cortex is the seat of lesion. A similar condition results also from lesion of the ganglia and internal capsule. In the latter case descending degeneration ensues.

The congenital and acquired defects may also lead to symmetrical or asymmetrical changes in the skull, either of the nature of enlargement or the reverse. When only one side of the skull is misshapen, it is mostly collateral with the cerebral defect.

The porous defects of the cerebral cortex may become cicatrised and closed, but their effects nevertheless continue.

These are only the leading points of a valuable monograph carefully prepared and illustrated.

DAVID FERRIER.

Exner on Cerebral Localisation. (Localisation der Functionen in der Grosshirnrinde des Menschen. Von Prof. SIGMUND EXNER. Wien, 1881.)

THE conclusions which Prof. Exner has arrived at from an analysis of the facts of disease as set forth in this work have already been noticed ('BRAIN' XI., p. 416). The methods which he has followed are here described in detail and copiously illustrated. Many reasons might be stated why the conclusions which Professor Exner has come to cannot be accepted as an accurate representation of the teaching of the facts of cerebral disease. His statistics of cortical lesions are vitiated by the inclusion of a large number of tumours, the symptoms in connection with which are obviously due to the indirect action on other parts than the immediate seat of the disease; and his doctrine of relative centres, founded only on the *frequency* with which lesions of these regions cause certain symptoms, cannot be regarded as based on conclusive evidence.

DAVID FERRIER.

A Treatise on the Diseases of the Nervous System. By JAMES ROSS, M.D. Two vols. J. and A. Churchill.

This is a work of very great value. It is very well written, and is as simple as the very complex nature of the subject admits of. Dr. Ross does not artificially simplify his subject by the lazy process of ignoring its difficulties. He considers it clinically, physiologically, pathologically, and does not fail to take into account the psychical side of what is physically nervous action. He recognises the great value of Herbert Spencer's work as bringing into fundamental harmony many superficially different phenomena of nervous diseases. There is no other work on Diseases of the Nervous System which covers so much ground, and is yet so harmonious in its several departments as is that of Dr. Ross. It is very difficult for a reviewer to do the author justice. It is too good a work for mere praise. We can only remark on some sections of it.

Quotations will be given as samples of the author's acuteness, originality, and power.

The work is divided into two books; the first is on the general pathology of the Nervous System, the second on its special pathology. The first chapter of book 1, about seventy pages, is on the structure and functions of the Nervous System. This is not the usual dry compilation from most recent writings on anatomy and physiology, although it gives most recent knowledge. Besides much originality, in the usual acceptation of that term, there is displayed throughout the chapter the originality of the man who thinks for himself. The headings of some of the sections will suffice to indicate the enlargement Dr. Ross gives to his topic; "Antagonism betwixt the Size of Units and Absorption of Nourishment." He writes, vol. i, p. 13, "that an increase in the size of a cell is followed by a relative diminution of material exchanges is readily proved by the fact that the surface which a large cell presents for the absorption of nourishment, does not increase in a degree proportionate with its bulk. The mass of a body increases as the cube, while the surface only increases as the square, of the dimensions. When, for instance, a cell has doubled its dimensions, its mass is eight times, while its surface is only four times, the original size. It is evident, therefore, that a small cell presents, in proportion to its bulk, a larger surface to its environment for the absorption of nourishment than a large cell, and, consequently, material exchanges take place more readily in the former than in the latter." Other headings, are "Differentiation of Structure, and Specialization of Function," "Integration of Structure," "Passage from the general to the special in both structure and function." The whole chapter is a splendid contribution to Neurology, and should be minutely studied. We quote the last two sections of it, partly because they give a slight summary of some important facts in the earlier sections. There is another reason. Many persons suppose that evolution is concerned only with the evolution of animal life, with what is commonly called Darwinism. Students of Spencer's System of Philosophy know better. Dissolution is the word Spencer has long used for the process the opposite of evolution, and is, therefore,

rightly applied to the results of disease of the Nervous System.

"*The Law of Evolution.*—We have now passed very rapidly and very imperfectly under review the fundamental laws of nervous structure and function. We have seen that the nervous system consists essentially of cells and fibres, that the cells are first small, round, and uniform; that they gradually become large and assume numerous processes; that the fibres are at first small fibrils, which together form larger fibres; that these become complicated by assuming an elastic sheath, and still further complicated by assuming a second sheath of a very special character. We have seen how the fibres come to be packed together to form white cords, and the cells to form small masses of grey substance termed ganglia; how the cords integrate to form thick masses of white substance and the small ganglia to form masses of ganglionic grey substance, and how this continuous process assumed a still more complicated form when some of the ganglia became subordinate, while others exercised superordinate functions. The whole of the intricate processes here described illustrate the one great law of evolution. That law may be described as a progressive integration, both of structure and function, during which there is a passage from the uniform to the multiform, from the simple to the complex, and from the general to the special. During the evolution of the nervous system of man, the fundamental portion is first developed. The nervous system of man is at first similar to that possessed by all animals which possess a nervous system, or, at any rate, all those which are sufficiently elevated to possess a spinal cord; but as development proceeds, the nervous system of man becomes gradually differentiated from that of an ever-increasing number of the lower animals, while still maintaining a general likeness to the nervous system of the higher animals up to the time of birth. This, then, constitutes the *fundamental* portion of the nervous system of man; but after birth the *accessory* portion, which up till this time only appears in a rudimentary condition, now undergoes progressive development, and the nervous system of man becomes gradually differentiated from that of all other animals. It will thus be seen that the fundamental portion is first de-

veloped, and that the superaddition of the accessory portion greatly increases the multiformity, the complexity, and the speciality of the human nervous system, and it is, consequently, the latest product of its evolution."

"*Law of Dissolution.*—We must now proceed to regard the phenomena of the structure and function of the nervous system from a new and opposite standpoint. We must watch the cells lose their processes, and from the multiformity of the caudate cells with numerous processes pass to the uniformity of the round cells destitute of processes; we must observe the fibres losing their medullary sheath, then their elastic sheath, and finally the axis-cylinder itself becoming disorganised, so that the nervous tissue gradually gives place to a simple and uniform connective tissue; we must observe accompanying this process a corresponding loss of function in which the complex movements that characterise health become difficult or impossible; in one word, we must trace the records of a process in which the progressive integration, during which the phenomena of structure and function, instead of passing from the uniform to the multiform, from the simple to the complex, and from the general to the special, manifest a reverse tendency, of passing from the multiform to the uniform, from the complex to the simple, and from the special to the general. The law which governs this process is the law of dissolution, and it is the great law which regulates the phenomena of disease of the nervous system, just as evolution is the great law which regulates its growth and development."

It is scarcely necessary to add that the phenomena of evolution manifested in the growth and development of the organism are exceedingly gradual and continuous, and consequently the operation of the law of evolution can very readily be traced. Disease being, on the other hand, often sudden and violent in its onset, striking at times at the fundamental, at other times at the accessory portions of the nervous system, now producing its baneful influence at one stroke, again acting fitfully, and only on rare occasions, in a gradual and progressive manner; it may be inferred that the operation of the law of dissolution can never be so clearly

traced amongst morbid phenomena as that of evolution in the development of the organism. Nevertheless there are some diseases of the nervous system which are gradual in their invasion and progressive in their course, and in them the operation of the law is clearly visible; and it is astonishing how glimpses of the law may be obtained, even when the disease is sudden in its onset and rapid in its progress. One important corollary may be drawn from what has been said; that as the *accessory* portion of the nervous system is the last to be developed, it is the portion which is most liable to become diseased. Several reasons might be given why this should be the case, but these will appear in the subsequent pages. It will suffice at present to say that the accessory portion, from the late period of its development, is less stable than the fundamental portion, and that its necessarily frail structure will render it more liable to suffer both from accident and the inroads of disease."

The first part of Book II. contains eight chapters dealing with affections of peripheral nerves, cranial and spinal. It is a very clear account; the difficult things are rendered simpler by help of many illustrations. In the case of motor nerves, both spasmodic and paralytic affections are considered. The next part is on diseases of the sympathetic system, under which Dr. Ross gives a very elaborate account of headaches. The description of migraine is very excellent. An account of exophthalmic goitre is given in this Part.

Diseases of the spinal cord are then considered; to the account of them we can give the highest praise. But the next Part, "Diseases of the Encephalon" is perhaps the most remarkable in the work. The "Anatomical and Physiological Introduction" shows the author's method well, and illustrates his grasp of neurology. It, like other parts of the work, contains many illustrations, and by them and the author's lucid exposition, a very complex subject is made comparatively easy. A careful account of the topography of the cerebrum and cerebellum is given, and of the plan of arterial supply of the different divisions and sub-divisions of the encephalon. Practical ends are never lost sight of; relations of the convolution to parts of the skull are stated and illustrated; orderly "dissections," if we

may use the term, of the brain are made, so that at post-mortem examinations exactness in recording the locality of lesions is rendered easy. The minute anatomy of the brain is dealt with; the researches of Lockhart Clarke, Meynert, Bevan, Lewis, and others being made use of. A brief account of some of Herbert Spencer's views on cognitions and feelings is given under the head of 'Functions of the Cortex of the Cerebrum.' The following, from Section 686—*Anatomical Substrata of Consciousness,* is very interesting, and, we believe, very important:

"It cannot be supposed that the large cells, with the distinct processes and definite connections found in the internal division of the third layer of the cortex, will readily undergo structural changes in the healthy adult, and it is much more probable that any new alteration of structure in the cortex will proceed from the small cells of the external layers of the cortex. The first layer may probably be regarded as an embryonic layer without any active nerve functions, and consequently the second layer and external portion of the third layer of the cortex, the cells of which do not possess definite connections with one another, or with nerve fibres, must be regarded as the areas excitation of which is attended by the highest consciousness."

J. HUGHLINGS-JACKSON.

Clinical Cases.

A CASE OF PAROXYSMAL CLONIC SPASM OF LEFT RECTUS ABDOMINIS, WITH SYMPTOMS POINTING TO THE EXISTENCE OF GROSS INTRACRANIAL DISEASE.

BY E. BUCHANAN BAXTER, M.D., F.R.C.P.

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THE following case is put on record for two reasons. First, because it presents a somewhat unusual combination of symptoms, one of which—rhythmic spasm of an abdominal rectus—I have never before witnessed or seen described by others. Secondly, because of the unexpected failure of post-mortem inspection to throw any light upon the nature and origin of the disease. So much has been accomplished of late years to increase the certainty and precision of our diagnosis in intracranial disease, so many cases have been recorded in which a conscientious study of symptoms has enabled a correct opinion as to the nature and situation of morbid changes to be formed, that we are naturally prone to overestimate our powers in this direction. In the present instance, though unable to arrive at any very definite judgment as to details, I had no sort of doubt that I should discover some gross lesion after death.

Mrs. D., aged 35, first consulted me on November 18, 1879, for a nasal discharge, associated with peculiar "nervous attacks."

She was married in April 1877, and was then in perfect health. In September of the same year, after a good deal of worry and anxiety, she began to suffer from certain nervous symptoms, which have steadily increased in intensity and frequency, without undergoing any alteration in their character. They are of two kinds: headaches, and what she calls "twitchings." Shortly after these symptoms made their appearance, a clear watery fluid, sometimes rather offensive,

occasionally tinged with blood, began to come away from the right nostril.

She is a small, fairly plump woman, with rosy cheeks and no appearance of ill-health. Her manner is quiet and sensible, the reverse of "hysterical;" intelligence above the average; makes no attempt to excite sympathy or to exaggerate her sufferings. Menses regular. Has never been pregnant.

On closer enquiry, the following points were ascertained:—

1. *Headaches.*—These occur almost daily. The pain is most severe, beginning at the root of the nose, and spreading round to the back of the head. Moreover, there is a constant fixed pain, like clavus, referred to a point on the left side of the occiput, just outside the insertion of the muscles of the neck. On deep pressure with the finger-tip, a small area of tenderness can be made out here. The headaches are not at all periodic; they are always worse during the daytime, and never keep her awake at night. She distinguishes between these constant headaches and what she calls "bilious attacks." The latter have recurred every fortnight or three weeks during the past twelve months. They consist of violent headache, aggravated by light, noise, and movement, associated with nausea and eructation of a bitter fluid. She never vomits at other times. She suffers a good deal from constipation, and the state of her teeth interferes with proper mastication.

2. *So-called "twitchings."*—Hardly a day passes without an attack, which may be brought on at any moment by the most trifling emotion, even by a knock at the door. She had one in my presence the first time I saw her, and I copy the description written down at the time. "The first thing one sees is a jerking of the left shoulder downwards and forwards, recurring at intervals of two or three seconds. The jerks are very sudden and forcible. They go on increasing in violence for several minutes, though, by straightening her arms, clenching her teeth, clasping her hands, and holding her breath, she evidently does her best to repress them. On stripping her, the jerking of the left shoulder, and indeed of the entire left half of the trunk, is seen to be due to a violent recurrent spasm of the left *rectus abdominis*. The spasm is extremely sudden and violent, quite like the spasmodic contraction of the diaphragm in hiccup. As it increases in frequency and intensity, the pectoral muscles become involved, and the left humerus is simultaneously jerked forward. No other muscles take part in the movement. At the height of the paroxysm, a patchy erythema with irregular, sharply-defined margins, makes its appearance on her forehead and chin, spreading gradually over the face and neck in a deep rosy blush. She then begins to shed tears and laugh just like a

patient in an ordinary hysterical attack. Even when she is at her worst, she makes obvious efforts to control herself, and answers questions sensibly. The paroxysm passes off as gradually as it came on." The duration of the paroxysm varies from ten minutes to a couple of hours. When it lasts for an unusually long time, she falls asleep from sheer exhaustion, but continues to twitch feebly even in her sleep. The fits of laughing and crying always occur in connection with the "twitchings."

3. *Discharge from right nostril.*—Of this she complains bitterly. On examining through the mouth, the upper jaw is seen to be crowded with decayed stumps, the gums round which are slightly ulcerated. The posterior wall of the pharynx is smeared with thin muco-purulent matter, which has found its way down from the naso-pharynx. The right nostril is pervious. No ulceration or disease of bone can be detected by examining with the rhinoscope or from the front. The discharge is only sometimes offensive. I never could perceive any unpleasant odour when I saw her.

There never has been any loss of consciousness or intellectual confusion. There is no failure of muscular power, no hyperæsthesia or anæsthesia anywhere. No spinal or ovarian tenderness. No impairment of taste or smell. When the condition of her eyesight was enquired into, she admitted that it had been failing for some three weeks. "Things look misty, and I seem often to see only one half at a time." On examination, the pupils were found equal and to react well to eyes. No squint. Incomplete but decided hemiopia in both light. Double optic neuritis.[1]

There is uniform soft hypertrophy of the thyroid body, first noticed two years ago, when the present illness began. Pulse 104, regular. Nothing amiss with heart, lungs or urine.

She has repeatedly sought medical advice during the last two years. Her symptoms have always been attributed to hysteria. No treatment, however, has had any influence, either on the twitching, or on the discharge from the nostril.

I was disposed, at the first glance, to agree with the view taken by those who had previously seen her. The absence of any failure of health and nutrition, the onset of the symptoms some months after marriage, their connection with mental worry, the almost invariable association of simply hysterical

[1] Shortly afterwards, I had an opportunity of showing her to Mr. Nettleship, who made the following note about her eyes: "V $\frac{R}{L}$ 7 Jäger. ∞ $\frac{12}{50}$. Colour-perception normal. Upper and outer quadrant of each f. v. either foggy or quite a blank. Neuritis of moderate intensity in either eye."

phenomena with the paroxysms of twitching—anomalous and even unique in my experience as these undoubtedly were—all pointed in this direction. But the discovery of the intra-ocular mischief speedily put me on the right track. Close observation, moreover, showed that the hysterical symptoms did not really form a part of the convulsive attack, but were engrafted upon it. They were due, as I convinced myself by subsequent investigation, to the emotional break-down consequent on the utter failure of the patient's vehement efforts to check or control the twitching. Again, her manner, the absence of any menstrual derangement, of any spinal or ovarian hyperæsthesia, were against the hypothesis of the functional nature of her malady.

Admitting, then, that the mischief was of organic origin, what was its nature and its seat? As regards the first question, the possibility of syphilitic infection was thoroughly considered, and after most careful examination of the patient and of her husband, rejected. It may here be added, that the absolutely negative result of anti-syphilitic remedies administered for a considerable time and in adequate doses fully justified this conclusion. The most likely hypothesis out of a number that presented themselves appeared to be that of some chronic disease of the body of the sphenoid or the neighbouring part of the ethmoid bone, leading to inflammatory changes about the optic chiasma and adjoining parts of the brain. Chronic mischief in this locality might account for the headaches, the neuritis and peculiar hemiopia, as well as for the discharge from the nostril. No theory of localisation would account for the twitching of the rectus muscle.

This, of course, was mere speculation. Of one thing I felt sure, that the symptoms were due to some coarse organic mischief, and that the prognosis was accordingly unfavourable. Of these two convictions, the second alone, as the sequel shows, turned out to be correct.

Here is a brief abstract of the subsequent notes of the case:—

January 6th, 1880.—No change in symptoms, save that her eyesight is failing. Ophthalmoscopic appearances the same as before, except for a small dotted hæmorrhage on the outer edge of the right disk. The headaches and "bilious attacks" as frequent and severe as ever.

Feb. 3rd.—Has not vomited since her last visit. Symptoms unaltered, save that for about a week her nights have been restless; she falls asleep and is roused by frightful dreams. The mucopurulent discharge from the right nostril is abundant, and occasionally streaked with blood.

May 25th.—Has felt a little better, on the whole, during

the last three months. Still looks plump and ruddy. Suffers a good deal from inframammary pain shooting through to the angle of the left scapula, with aching of the left arm. This is clearly due to over-fatigue from the frequent and violent twitching.

June 15th.—Has had two attacks of vomiting since her last visit. One was extremely severe and lasted for fourteen hours. Eyesight failing. No material change in fundus oculi. For the first time, I find her looking rather pale and tremulous. She ascribes this to the agonising and almost continual headache.

August 4th.—Vomiting has been very unfrequent. Otherwise no material change. No signs of atrophy of disk as yet.

Sept. 28th.—Still looking rosy and well. Sleep disturbed for a fortnight. She sleeps for five minutes and wakes up under the impression that she has been asleep for hours. The headaches have often been attended with severe vomiting. Nasal discharge persists. So does the thyroid enlargement.

Oct. 27th.—In a very tremulous state. Her headaches have been aggravated, during the last eight days, by frightful neuralgic pain in the left eyeball. The conjunctiva of this eye is intensely injected over its outer half. Eyeball tender on pressure; no increase of tension. Pulse regular, 100. Other symptoms unaltered.

Nov. 25th.—Looks really pale and ill. Outer half of left eyeball still much injected. Can just make out 19 Jäger with either eye and with a limited portion of each retina. Decided photophobia. Olfactory sense continues normal. No change in ophthalmoscopic appearances. Has not vomited for four weeks, though the pain across the temples and behind the root of the nose is as bad as ever.

This was her last visit to me. She ceased to be able to leave her home, where she was under the care of Dr. Poole of Sidcup, to whose courtesy I am indebted for what I know of her subsequent progress and for the opportunity of examining her after death.

She appears to have become steadily thinner and weaker, and to have suffered constantly from pain in the head and vomiting. She did not take to her bed, however, till about a fortnight before her death, which took place on the 29th of January, 1881. Three days before she died she had general convulsions, which recurred frequently and terminated in coma. Her intellect remained unaffected to the last, although a sudden change was observed in her frame of mind. Whereas she had previously, amid all her sufferings, remained cheerful and resigned, about ten days before her death she became pro-

foundly melancholy, taking the most gloomy views of her religious condition.

A word as to treatment. It has already been stated that a full trial was given to the iodide of potassium in large doses, and to mercuric chloride. They produced no effect. She took, for the relief of her symptoms, bromide of potassium, occasionally chloral hydrate and hydrobromic acid. The only relief to her headaches was given by small blisters kept open with Ung. Sabinæ. The decayed stumps were all extracted from her upper jaw, and she employed a nasal douche of potassic permanganate. This prevented the offensive smell of which she complained, but which was never very marked.

At the post-mortem examination, made by Dr. Poole and myself, the head only was opened. The body was thin, but not emaciated. No signs of decomposition. The bones of the skull appeared to be more thick and dense in texture than usual, offering considerable resistance to the saw. The interior of the skull, the brain, and its membranes, were very carefully examined in a good light, and nothing in any way abnormal was discovered. With bone-nippers I made my way into the cavities of the sphenoid and ethmoid, without finding any evidence of disease. It ought, perhaps, to be added, that no appearance was anywhere met with calculated even to excite suspicion and call for more minute examination.

The negative result of the inspection was a surprise to me. The experience of others may, perhaps, contribute to the explanation of what remains to me inexplicable, and it is with this hope that I publish the case.

A CASE OF TUMOUR IN THE MEDULLA OBLONGATA AND PONS VAROLII, WITH REMARKABLE PARALYTIC SYMPTOMS.

BY J. M. HOBSON, M.D., EDIN.

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[This report is only a fragment of what it should have been, but I trust that its intrinsic interest will justify its publication.]

THE case was admitted into the North-Eastern Hospital for Children in Hackney, on February the 27th, 1880, under the care of Dr. Sansom, with whose kind permission I now publish it from the notes which I made at the time.

Benjamin L., aged 2 years, was quite well and able to run about and talk till *nine weeks before admission.* At that time he began to waste, and to have difficulty in swallowing. *Five weeks* later he began to have some impairment—I was told by his father—in the movements of the left arm, and the left upper eyelid began to droop. Then this side of the body was said to get better, and the corresponding parts on the right side became affected in the same manner. But previous to the affection of the arms the legs were effected. The date of this, however, is uncertain, as the fact was obtained more than a year after the child's death.

On admission the child was certainly a most remarkable object. His body was very thin and his head very large, his face expressionless, and saliva dribbled from the corners of his mouth. He had no power of articulation, considerable difficulty in swallowing, and frequently made a noise in his throat, as if some saliva were there which could not be got rid of. The right eyelid *at times* drooped, but not constantly, and further observation showed that this was due to contraction of the orbicular muscle. There was no apparent squinting or inequality of the pupils. From time to time there were clonic contractions of the right side of the mouth. Not only was he incapable of standing when supported, but he was unable to sit upright in bed, and, when unsupported

and not lying back, his body was bent forward. The position of his head varied; sometimes it was drawn much back and towards the right shoulder. While the right arm was lying unused it was quiet, but any attempt at movement excited a *rhythmical jactitation* of the limb. At some of these times the whole body was jerked.

The optic discs were normal. The temperature was normal. The bowels were costive, but everything was passed under him.

On March the 5th I made the following note:—"It is clear that the left side of the face is paralysed. Saliva dribbles from either side. The movement of the head [above mentioned] can be induced by creating a necessity for its support by the child's own muscular effort [i.e. by removing all artificial support from the head]. Three times to-day, when the spasm was so evoked, the right sterno-mastoid became rigid and the chin was moved to the left; once the head approached the *right* shoulder, but on the other occasions this was not the case. There seems to be some tendency for the *left* arm to be jerked during voluntary movement. The rhythmical jerking of the *right* arm is very free."

On March the 9th I noted:—"The legs are sometimes drawn up stiffly. The left arm certainly jerks on voluntary movement. Once this evening the chin was moved to the *right* side, and at another time there was strong contraction of the right side of the face, and the head was drawn back."

On the night of March the 15th the patient was so ill that it seemed he could not survive till morning. However, on the 17th he was no worse, and could swallow better than he had been able to do for some time past. Both arms would jerk when he attempted to get hold of anything, and the fingers of the right hand were got round any object with difficulty. Ever since admission he had been very irritable, making a little cracked squealing noise when not pleased, and, at such times, only the right side of his face would be used.

He died on March the 22nd, a little over three weeks from admission. He had been getting lower for some time. When last observed, there was no obvious rigid drawing up of the legs. No marked changes were observed in the optic discs before death.

I have, unfortunately, no note at the time as to sensation. It would clearly have been impossible to gauge this with any precision.

Necropsy.—There was much subarachnoid œdema. The ventricles were filled with fluid to distension. No structural lesion was found in the brain till the medulla was inspected, which was *bulbous*, indeed—twice the normal size. On section,

a tumour was found occupying in an irregular manner the central and posterior part of the upper half of the medulla and, in the pons, being confined almost entirely to the left side (see Figs. 1–4).

Microscopically it was found that the growth was true tubercle, well showing, although much was caseating, the characteristic giant-cells with small round cells and trabecular framework (see Fig. 6).

Very unfortunately the sections were, in great measure, a failure, for, between their being cut and their being mounted, the majority fell to pieces, and the result is that I am not able to show a single good section of the medulla through the mass of the tumour. However, I have enough material to indicate in broad outlines the pathological anatomy of this case.

I give on the next page some drawings taken from the more successful specimens:

Fig. 1 shows the space occupied by the tumour at the middle of the pons. It reaches to the floor of the fourth ventricle, and will cut off, on the left side, all the vertical fibres, except a few anteriorly, and on the right side, a few fibres near the middle line.

In Fig. 2, a section through the highest planes of the medulla, none of the vertical fibres have, apparently, escaped on the left side, while the tumour still transgresses a little the middle line.

Fig. 3 shows the tumour still farther invading the right side. This section passes through the olivary body; but, unfortunately, the whole of the anterior pyramid is wanting on the left side. Yet this is not of so much consequence, for we know that those descending fibres have already been cut off above.

In Fig. 4 the tumour occupies nearly equal portions of the medulla on either side. The olive has only been indicated on one side, as the section was too thick on the other side for it to be traced. Neither can I say which is right and which left side. It will be noticed that the tumour encroaches very much on the fourth ventricle. Evidently, the two halves of the medulla have been forced apart from behind by the tumour as by a wedge, hence the displacement towards the middle line of the outer end of the olive.

Below this section the tumour must have rapidly thinned out, as in places where the olive is still well developed the tumour either remains as a very small patch near the middle line, or is entirely absent.

In mid-pons the tumour is getting smaller, but I have no sections to show its vanishing point above.

One might expect that in this case there would be atrophic

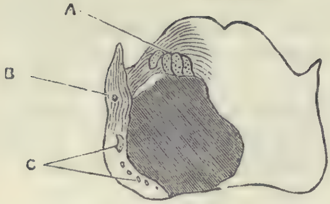

Fig. 1.

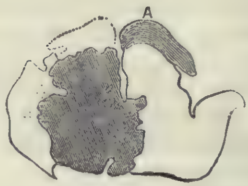

Fig. 2.

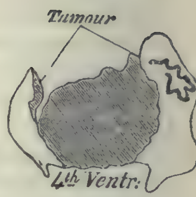

Fig. 3.

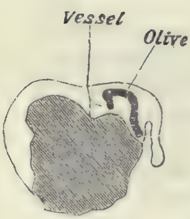

Fig. 4.

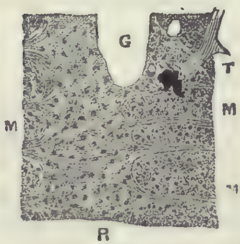

Fig. 5.

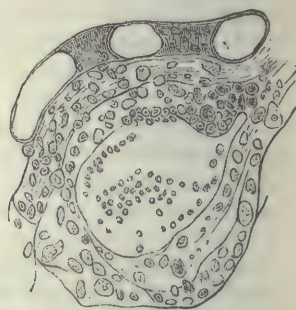

Fig. 6.

FIG. 1. Transverse section through Pons at the level of emergence of fifth nerves.

A. Bundles of descending fibres of crusta.
B. Great root of fifth nerve emerging.
C. Outlying portions of tumour.

FIG. 2. Transverse section through highest planes of medulla.

A. Lowermost fasciculi of superficial fibres of pons included in the section.

FIG. 3. Transverse section through middle of medulla. The left anterior pyramid is wanting.

FIG. 4. Transverse section through medulla below former. Part of the section is wanting behind on the right of the figure.

FIG. 5. Transverse section through edge of tumour at its lowest extremity, showing infiltration of the surrounding structures with leucocytes, magnified 37 diam.

T. Tumour.
R. Raphé with transverse bundles of fibres proceeding from it on either side.
M, M, M. Transversely cut bundles proceeding to ant. cols. of cord.
G. Gap for vessel.

FIG. 6. From edge of tumour, magnified 130 diam:, showing a giant cell imbedded in delicate stroma and smaller cells of varying size. At its apex the great cell is apparently budding off a number of small cells.

changes in the descending fibres of the medulla, but there were none present; there does not seem to have been time enough for their development.

Let us now try to trace the connections between the nervous symptoms and the structural lesions.

In the first place, as we have seen, the difficulty of swallowing was the earliest symptom of nerve lesion. This fact, taken with the other that the tumour reached the periphery of the medulla at the fourth ventricle only, seems to point to the new growth having started there.

The paralyses separate themselves into two classes—A. of cranial nerves—B. of spinal nerves.

A. The order in which the cranial nerves were affected was, (1) those presiding over deglutition, viz.—the vagi, accessories, glossopharyngeals and hypoglossals. The symptoms were, impaired deglutition, loss of speech, and tonic contractions of the sternomastoids and trapezii. (2) The left facial. The symptoms were, first, drooping of the left upper lid, which was probably spasmodic, and, subsequently, paralysis of the left side of the face. (3) The right facial. The symptoms were, occasional drooping of the right upper lid, which was spasmodic, and occasional clonic contractions of the right side of the mouth.

A nerve may have its functions interfered with in one or more of three ways. Damage may be done to (1) its nucleus or nuclei of origin; (2) the strands connecting its nucleus or its roots with the cerebral hemispheres; or (3) its roots proceeding peripherally from the nucleus.

The nuclei of the vagi, cranial accessories, and glossopharyngeals are in the floor of the fourth ventricle, or just beneath it. At the level of Figs. 3 and 4, these nuclei should exist within a small area extending outwards from the middle line; but it is here that they are quite destroyed, and also that all communication with the hemispheres is cut off. Lower down, however, where the central canal is just closed in, and the tumour is just thinning out, the lower extremity of one of the vago-accessory nuclei (the tuberculum cinereum) is quite free, and fibres of the accessory can be traced outwards and forwards from it. Close beside this latter nucleus, on its outer side, is a band of vertical fibres (the "solitary fasciculus") which ascending, according to Meynert,[1] from *crustal* fibres, probably from just above the decussation of the pyramids, crosses the raphé, and distributes its fasciculi to the rootlets of the vagus, accessory, and glossopharyngeal nerves. From the vagus and glossopharyngeal these fibres

[1] Theodor Meynert "On the Brain of Mammals." 'Stricker's Histology,' vol. ii. Syd. Soc., 1872.

would be quite cut off, but the accessory would receive contributions from it. The continuity of the *spinal* rootlets of the accessory would of course be quite free.

"Opposite to the vago-accessory nucleus, and enclosed by the transverse sections of the posterior division of the medulla oblongata, is situated the *anterior* column of origin of the *lateral mixed system* [glossopharyngeal, vagus, and accessory] in the form of an oblong nucleus lying at a distance of three millimetres from the grey substance of the central cavities. . . . This *anterior nucleus of origin* of the lateral system represents in the medulla oblongata the *processus lateralis* of the anterior cornu of the spinal column. The former allows the upper, the latter the lowermost, fasciculi of origin of the lateral system to arise from it as the accessory nerve."[1]

This nucleus is distinct from the *tuberculum cinereum.* We shall presently see the significance of this. The nuclei of the hypoglossal nerves lie in planes mostly below the level of the tumour, and, though some of their roots will pass through the tumour, many will be quite free from it. The *central* connections of the nuclei are through the raphé and the anterior pyramids of the opposite side. As the fibres of the right pyramid only are quite (if that) cut off above, the left pyramidal fibres of the nuclei will be continuous, in part at least, with the hemispheres; and as, moreover, there are commissural fibres between these two nuclei of concerted action, the central connections of both will be in part retained.

The facial nerves.—On the *left,* the tumour, at the level of Fig. 1, would have entirely obliterated the facial on that side. The area of the *right* facial nuclei and roots is throughout intact, but their central links of connection suffer partial interruption. These links are threefold. (1) From the common nucleus of the sixth and facial which, after entering the raphé as fibræ rectæ, "run obliquely downwards towards the medulla oblongata, curve round [from the posterior to the anterior part] at the lower border of the pons, and run with the pyramids into the crus cerebri"[2] of the opposite side. (2) Fibres which enter the raphé directly from the facial *root* and decussate. (3) Meynert says that the central fibres from the inferior nucleus of the facial "may connect the nucleus through the raphé with the crus cerebri." Thus all these fibres entering the *right* half of the pons from the *left* of the middle line run the risk of being invaded by the tumour growth.

Sixth nerve.—It does not seem possible for the nucleus of the *left* nerve to have escaped destruction, while the *right*

[1] Meynert, op. cit. page 506. [2] Ibid. p. 491

nerve would, like the facial, run the risk of interference with its central fibres.

Having acquainted ourselves with the foregoing anatomical facts, we are in a position to follow up some of the symptoms to their origin in the morbid processes.

First, with regard to the areas supplied by motor filaments from the eighth pair of cranial nerves. The destruction of the centres of the vagi and glossopharyngeals and, partly, of the *cranial* accessories will amply account for interference with deglutition and phonation without enquiry as to how far the hypoglossals were at fault. But what caused the loss of speech? The child had begun to speak but had left off again, and I have neither note nor recollection of his ever making the least attempt at conveying his ideas, save by the "little cracked squeal" above mentioned. Yet we have not found enough in the condition of the hypoglossals to account fully for this. I do not remember his ever being told to "put out his tongue." Could it be that the difficulty in using his larynx and lips and tongue, and his generally demoralised state, made the attempt at employment of the little speech he had acquired too irksome?

The destruction of the upper part of the anterior accessory nucleus, with infiltration of its structure by the *advancing border* of the growth lower down, might well lead to an "irritation" of its spinal portion, which would be expressed by occasional spasmodic contractions of the sterno-mastoids and trapezii.

The initial but transient drooping of the left upper lid, mentioned by the father, and the subsequent *spasmodic* drooping of the right upper lid observed by me, the occasional twitching of the right side of the mouth, and the permanent paralysis of the left side of the face, are all easily accounted for. At first the tumour only "irritated" the left facial nerve, but, as it grew, destroyed that nerve, and began the same operation on the opposite one.

As to sixth nerve. The left nerve must have been wholly paralysed, yet no squinting was observed. But this is just one of those things that might escape notice if not specially looked for, and specially looked for it was not.

B. *Paralyses of spinal nerves.*—On the left side of Fig. 2 we find that all, or nearly all, the fibres of the anterior pyramid have been destroyed—the imperfection of the specimen does not allow us to say that a very few of the anterior fibres did not escape—while in Fig. 4, some portion of the anterior pyramid remains to each side. All we can say for certain is, therefore, that only a very few fibres of the *left crusta* could have reached the *right* half of the cord. On the other hand,

although portions of the anterior pyramids do, as I say, remain in Fig. 4, all their structure to the left hand of the small vessel indicated in the figure is much infiltrated by cell-growth. Hence the left half of the cord must also have lost much of its connection with the opposite crusta. This is plain, although we cannot tell which is right and which is left in the figure.

But first of all, this question presses for answer. If the child had lost all his anterior pyramid on the left side, how was he able to move his right arm at all?

It is in man that the crusta attains its highest development, and this is always commensurate with the development of the cerebral hemispheres in animals. This is not the case with the tegmentum. Compare the figure of the transverse section of the calf's medulla given by Van Der Kolk in his treatise on the Spinal cord and Medulla,[1] with the one he gives of the medulla of Man. It will be seen that in the former the great mass of the transversely cut fibres belong to the tegmentum, while the crusta is represented by the feeble bundle of the anterior pyramid. Meynert[2] divides the tegmental fibres in the human medulla into two groups; (1) "the region of the *anterior column* forming the territory adjacent to the raphé, that is, bounded laterally . . . in the oblongata by the roots of the hypoglossus;" (2) "the region of the *lateral column* extending from this boundary as far as the ascending roots of the fifth," i.e. to the outer border. Meynert calls these fibres motor, but insists on their functional separation from those of the crusta. He points out that these latter proceed uninterruptedly from the corpus striatum to their destination in the lateral columns; whereas the former, proceeding from the optic thalami and corpora quadrigemina, become interlaced with the arcuate system of the sensory tracts, being brought into more intimate relation with them, and thus with the cerebellum, through the agency of scattered nerve corpuscles.[3] However, it is still uncertain what the function of these fibres is. May it have been that, failing its anterior pyramidal fibres, the right half of the cord was supplied with motor filaments through some of these lateral tegmental bundles of the same side? For a few would have escaped on either side of Fig. 4.

The legs were quite paralysed to voluntary movement, and the trunk nearly so. From soon after admission, onwards, their was rhythmical jactitation in both arms on any attempt at voluntary movement. A similar movement had been seen to occur in the trunk, along with that of the right arm (see p. 2). This kind of movement was exceedingly like that of

[1] New Syd. Soc., 1859. [2] Op. cit. page 467.
[3] Op. cit. pages 452, 474–78.

Multiple Sclerosis. Could it have been that the cause in each disease was the same, namely, a blocking of *some* of the motor fibres to the several muscles, but not of *all?*

As there was no coma, the effusion into the ventricles must have been gradual, and have allowed the cerebrum to accommodate itself to the pressure. Was the cause of the effusion the obstruction of the communication between the fourth ventricle and the arachnoid cavity of the cord?

The new growth was essentially infiltrating. At its periphery the proper structure of the organ can be well seen, with leucocytes lying amongst it. There was some difficulty in defining the outline of the tumour in Fig. 4; indeed, the outline given is necessarily a rough one, so much was the surrounding tissue infiltrated. In the sections from which Fig. 5 was taken, and in sections just below it, the appearances suggested, at first sight, secondary degeneration, but closer inspection showed them to be due to infiltration at the periphery of the growth. Sections a little lower were quite free from any excess of leucocytes.

The only spot where the tumour seems to be clearly separated from the surrounding normal tissue, is from the bundle A, in Fig. 1, to the emergence of the fifth nerve, i.e. along the line of contact with the superficial band of cerebellar fibres of the pons.

ARRESTED GROWTH OF THE LEFT ARM AND LEG IN AN EPILEPTIC PATIENT. LOCALISED ABSENCE OF GREY MATTER IN THE MOTOR AREA OF THE OPPOSITE CEREBRAL HEMISPHERE.

BY M. D. MACLEOD, M.B., EDIN.

Assistant Medical Superintendent, Cumberland and Westmorland Asylum, Garlands, Carlisle.

J. F., aged 36, admitted March 10th, 1875, from Whitehaven. Is stated to have been imbecile, epileptic, and a cripple from birth. Was sent to the Asylum because he became noisy and violent, and could not be controlled at home.

On admission, he was excited and somewhat violent. He was an imbecile. Could, as far as his physical deformity allowed him, attend to his personal wants, and when in the humour could answer simple questions.

His stature generally was small (60 inches). The right side of his body was muscular and proportionately developed. The left arm was undeveloped and useless functionally. Its various parts, bones, muscles, fingers, nails, &c., were all present, but much less in size than the corresponding parts on the right side. This arm was adducted, flexed at the elbow and wrist, and the thumb rigidly flexed on the palm. The left leg was two inches shorter than the right. It was flexed at the knee, slightly so at the hip, and the heel was drawn up. All the bones were smaller and shorter than those of the opposite extremity. Some of the muscles arising from the pelvis, such as the glutei, the rectus, and the hamstrings, were fairly well developed, but below that the muscles were quite undeveloped. The foot was shorter and smaller than the right foot. He could move the limb at the hip-joint, but it was of no use to him in locomotion. He moved from place to place by hopping on his right leg, which he could do with great dexterity and endurance. Those limbs were not merely rudimentary limbs, but limbs whose growth was arrested in early life. Their bones had ossified and grown to a certain size, but the muscular tissue, from want of use, had not grown. The skin of the limbs was sensitive.

He took two or three epileptic fits in each week. The first stage of the fit was longer than usual. He uttered loud cries for twenty or thirty seconds before being convulsed. There was no convulsion of the left arm, but the muscles attached to the pelvis on the left side were more or less convulsed during a fit. He remained in the Asylum till the 9th of November, 1881, when he died of pneumonia of both lungs and epilepsy.

Post-mortem appearances.—The body was in fair condition as to fatness. The hair on the head was thick and coarse, as was the beard. The body generally, except the left arm and leg, was covered with strong thick hair. The state of the left arm and leg is described above.

Head.—Scalp thick; skull-cap thick, dense, hard; outer table much developed. The dura mater was thick and tough, its vessels engorged. On removing the dura mater a distinct depression was apparent on the upper surface of the right hemisphere in the parietal lobe. The pia mater was congested, and not so easily separable from the convolutions as usual. The brain weighed $38\frac{1}{2}$ oz.; the right hemisphere $15\frac{3}{4}$ oz.; the left hemisphere $17\frac{3}{4}$ oz., and the cerebellum and pons 5 oz. The convolutions of the right hemisphere were atrophied generally, more markedly in the frontal and parietal lobes. Those of the left hemisphere were better developed, and had a more plump appearance. The depression on the surface of the right parietal lobe was in those convolutions at the upper end of the fissure of Rolando. In the marginal line it was two inches in extent from before backwards; on the outer, one and a quarter inches from above downwards, and five-eighths of an inch on the internal aspect. This depression was filled with a loose connective tissue, which was incorporated with the pia mater, and which adhered to the bottom of the depression. The posterior end of the first frontal, the upper ends of the ascending frontal and parietal convolutions, and the posterior part of the supra-marginal lobule, as far as the calloso-marginal fissure, formed the margins. At the margins of the depression the convolutions were smooth, rounded off. There was no grey matter in the bottom of the depression, the grey matter of the convolutions ending at its edges, the connective tissue being joined to the white matter of the hemisphere. There were no tracts of atrophy apparent proceeding from this depression, nor were either the ganglia, the crura, nor medullary tracts smaller on one side than the other. The left hemisphere presented no abnormality. The brain on section was firm; much congested. The cerebellum and pons were firm, congested, dark in colour.

Both lungs were in a state of red hepatisation, passing into

grey. With that exception the other organs and viscera were healthy.

Remarks.—The general impression given by the appearance of the cerebral lesion was that the brain-substance at this spot had been destroyed at birth or during very early life, probably by an injury causing effusion of blood, which became organised in time into the loose tissue which filled the depression. That the injury took place during very early life is rendered more probable by the fact that there was no trace of fracture or depression to be seen in the skull-cap.

It is of interest to note that the portion of convolutions absent or destroyed corresponds closely to the area in which Dr. Ferrier places the centres for the various complex movements of the arms and legs. These are the centres numbered by Dr. Ferrier, 2, 3, and 4.[1]

The question of connection between the ungrown limbs and the cerebral lesion naturally arises. I incline to think the cerebral lesion was the first condition produced. This caused motor paralysis of the limbs, and arrestment of growth from want of use. Probably, too, the lesion was of such a severe character as not to permit of compensatory motor action from co-ordination with the centres of the opposite hemisphere being established. A lesion of such an extent in the motor area might also produce a general nervous instability, and be the cause of the epilepsy.

All this, however, is a matter of speculation, as no accurate history of the man's early life could be obtained. The coincidence of the arrested growth of these limbs and the position of the cerebral lesion, as it more or less corroborates the results obtained by physiologists in their experimental researches into the motor functions of the cerebral surface, is, to say the least of it, somewhat remarkable.

[1] See 'Functions of the Brain,' pp. 141–2, 305.

PATHOLOGICAL CONTRIBUTIONS ON THE COURSE OF THE OPTIC NERVE FIBRES IN THE BRAIN.

BY J. DRESCHFELD, M.D., M.R.C.P.

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WITH the advance made in the study of cerebral localisation the question as to the total or partial decussation of the optic nerves in the chiasma, their further course in the tract and within the brain, and their connection with a visual centre situated in the cortex of the brain, has again come to the fore, and been made the subject of numerous observations and investigations.

The subject has been studied anatomically, by tracing the course of the fibres, and their connection with the cerebral ganglia and with the cortex; it has been studied experimentally on animals in various ways; and lastly, we have the clinical observations and the study of the lesions found after death in such cases.

Of all these different lines of investigation the clinical observations and the pathological revelations have, so far, given the most concordant results, and, while I hope shortly to consider the whole subject, in reference to these several lines of research, I wish merely in this short notice briefly to describe two cases, with post-mortem results, which have an important bearing on this subject.

The history of the first case is partially given in the 'British Medical Journal,' Aug. 7th, 1880; the patient has since then died, and I am now enabled to give the whole history of the case, with the results of the post-mortem examination, which entirely confirmed the diagnosis made *intra vitam.*

The patient was seen by me for the first time on April 3rd, 1880, at the Convalescent Hospital at Cheadle, when the following notes were taken by Mr. Stanwell, then resident Clinical Clerk.

History.—N. H., aged 40, joiner, married, with no history

of syphilis, gout, rheumatism, or alcoholic excesses, had been ill for two years, and unable to work during the last eighteen months, suffering from giddiness, headache, double vision, with numbness, weakness and trembling of the left side. He had not suffered from vomiting. The bladder and rectum had not been affected. A week before admission, he had one morning before rising three fits of distinctly epileptic character; his memory had recently become impaired, and he suffered from occasional attacks of headache, associated with febrile symptoms.

State on admission.—The patient is strongly built, sallow, fairly nourished; he has an anxious expression, and a hesitating manner. The left side of forehead is relatively smooth, while the right side is habitually wrinkled; the left palpebral fissure is slightly the larger (he can, however, close the left eye). The left angle of the mouth is rather lower than the right; the movements of the left side of the face are less extensive. The left upper limb is colder and paler, and the left forearm measures a quarter of an inch less in circumference than the right. The grip of the left hand is weaker, and the whole limb is involuntarily and frequently jerked about. These spasmodic movements affect the forearm and arm, but not the hand or the fingers; they are increased by effort or attention, and especially when any movement is attempted by the right hand (as in writing) or by any reflex excitement of the right side (such as tickling the right foot). The movements cease during sleep. The left leg is weaker than the right; it also is the seat of involuntary spasmodic movements, less in extent, but of the same character as in the left arm, and equally increased by exciting reflex contractions. The patient can walk, but in doing so drags the left leg. The patellar tendon reflex is considerably increased on the right side in both upper and lower extremities. The electric contractility is normal on both sides.

Sensation.—There is total left hemianæsthesia, exact in extent, both before and behind; the sensibility to contact, to pain, and to temperature being much diminished. The muscular sense is normal on the left side, and the patient distinguishes well different weights supported on the left hand; he can stand and walk with closed eyes. The pin-punctures bleed equally on the left as on the right side. The pupils are equal and active. There is no nystagmus or ocular palsy, but there is exact and total left hemianopsia of each field of vision. (The term "hemianopsia" is used, as Hirschberg proposed, and Mauthner and others have since adopted it, to relate to the absent field of vision, applying the term hemiopia for the insensible part of the retina—the patient

suffered, therefore, from homonymous left hemianopsia or right homonymous hemiopia). The central vision is normal; the fundi are normal; the veins of the fundus somewhat large, those in the left somewhat tortuous. Smell and taste are normal. The ticking of a watch is heard only when the watch is in contact with either ear (the deafness is, however, of old date). The urine is normal.

Diagnosis.—The symptoms of the case were so typical and well-marked, and corresponded so well with what had been seen in other cases, published, with post-mortem accounts, by Gower, Hughlings-Jackson, Hirschberg, and myself, that the case was looked upon as one of organic lesion, most likely a tumour, implicating the thalamus, the posterior part of the internal capsule, and the optic tract of the right side of the brain.

Progress.—The further progress of the case is chiefly interesting on account of the influence of the electro-magnet, the repeated application of which produced a permanent amelioration of the anæsthesia, especially of the left arm and neck, without, however, in any way interfering, even temporarily, with the hemianopsia. (For a further account of this stage in the history of the case I may refer to the account in the 'British Medical Journal' above quoted.) During the further progress of the case, there was chiefly noticed an impairment of the mental condition of the patient; he wandered about the wards at night, disturbed the rest of the patients, and was therefore removed to the Manchester Infirmary on May 26th. For a short time he remained in the same condition. Soon, however, his mental condition became worse; he became delirious and at times violent; the hemiparesis also became worse, so that he was almost completely hemiplegic; the involuntary movements ceased, the hemianopsia remained the same, and the hemianæsthesia (as far as the patient's condition would allow an opinion to be formed) remained in the same improved condition in which we found it after the repeated application of the electro-magnet. The patient gradually grew weaker, and died on Sept. 5, 1880.

The further symptoms which had been observed during his stay in the Infirmary (namely the gradually increasing weakness of the left arm and legs, the cessation of the involuntary movements and the appearance of the mental disturbances) were believed to be due to an extension of the tumour forward, implicating more of the motor tract in the internal capsule, and to softening in the neighbourhood of the tumour.

The post-mortem examination was made by Mr. A. H. Young, Pathologist to the Infirmary, to whom I am also indebted for the accompanying sketch (Fig. 1). All the organs except the

brain were firm and healthy, and here again the right hemisphere only was found affected.

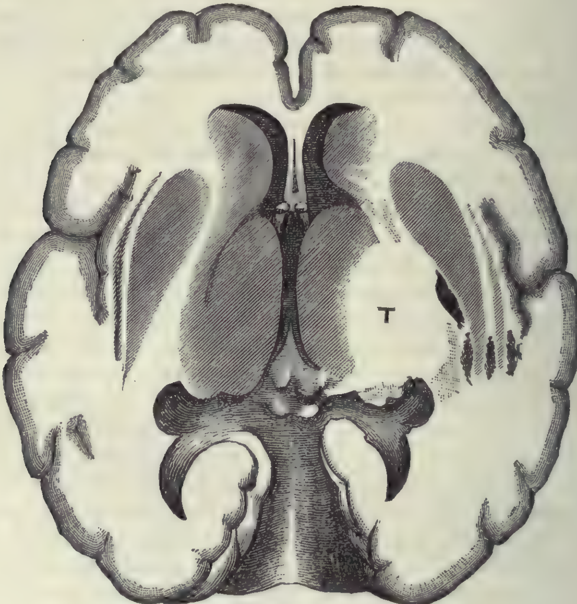

Fig. 1.

The right cerebral hemisphere appeared on superficial examination to be slightly more prominent than the opposite hemisphere, but there was no perceptible flattening of its convolutions. On transverse section of the brain there was found a tumour, diffuse in its distribution and in no way circumscribed or encapsuled, hence the determination of its exact limit was impossible. The main mass of the tumour, however, was situated in the posterior part of the internal capsule, extending from this as a centre to the optic thalamus, the lenticular nucleus, and the anterior of the corpora quadrigemina of the right side; all of which were infiltrated with new growth, and felt somewhat denser than normal. The anterior corp. quadrigem. was distinctly enlarged. The optic thalamus and lenticular nucleus were partly replaced by tumour-masses, partly simply pushed aside, so that their distal margins were more unduly separated than those of the corresponding opposite bodies (see Fig. 1). On examining the base of the brain, the right optic tract, just where it crosses the crus, and where itself becomes hidden by the gyrus hippocamp., was found flattened, and somewhat softer than the corresponding left tract.

The tumour was not, however, solid throughout. There was an irregular cavity in its interior, which was opened by the last incision made to expose the tumour, and it appears in the sketch as an oval opening to the inner side of the lenticular nucleus near the posterior end of the latter structure, and involving also the posterior extremity of the claustrum; the substance of the brain was softened and presented a loose reticular appearance.

Microscopic sections of the tumour showed it to be of a sarcomatous nature. It consisted chiefly of round cells, with a reticulum of fine fibrillæ, a few spindle cells, numerous blood-spaces, and embryonic blood-vessels.

The medulla and spinal cord, examined after hardening, showed the characteristic descending scleroses. I have recently again examined the sections of the cord, with the view of finding the sclerosed patch seen by Gowers in a case where there was anæsthesia, and which is situated anterior to the lateral pyramidal tract, but I found in this case no sclerosis on the spot indicated.

The symptoms observed during life are, as will at once be evident, satisfactorily explained by the situation of the tumour found in the brain. The tumour chiefly and primarily occupying the posterior part of the internal capsule explains the hemianæsthesia. The paresis and involuntary contractions observed during the first course of the disease are chiefly due, most likely, to an irritation of the motor fibres in the anterior part of the internal capsule, from the growth and extension of the tumour anteriorly. This state of irritation gave place to a state of paralysis, and hence we notice, during the later progress of the case, the disappearance of the involuntary movement, and the increase of the paralysis of the left side.

As for the hemianopsia, this can be accounted for in more than one way. We have noticed that the right optic tract was softened and flattened; though microscopically examined, it presented no decided changes. This would, perhaps, be enough to account for the hemianopsia; though we know, on the other hand, that cerebral nerves will often, by slow pressure, become compressed, without, however, becoming impaired in their function. The thalamus opticus, again, was found affected to a very great extent; and this, especially when considered with the second case to be described, and with some recent cases recorded,[1] might in itself be sufficient to account for the hemianopsia. Lastly, the right anterior ant. corp. quadrigem. was found enlarged and infiltrated

[1] (Pflüger, 'Bericht der Berner Augenblick,' 1818; Remy, Nothnagel, Topische Diag., &c., p. 231. Hughlings-Jackson and Gowers, Ophthalm. Hosp. Rep., vol. viii., p. 330.)

with new growth, and its connection with the optic tract is sufficiently well known. While thus the case does not teach us much as regards the course of the optic nerves in the basal ganglia, it offers an important support to the theory which advocates partial decussation of the optic nerves, and from this point of view deserves to be placed on record.

The second case I wish to put on record is one in which the lesion which produced the hemianopsia consisted of a small hæmorrhagic focus situated in the posterior portion of the thalamus opticus, without in any way affecting either the tract, the corp. geniculata, or the corp. quadrig. For the purposes of localisation, this case, therefore, is of great importance.

In describing the history of the case and post-mortem appearances I can be very brief.

Mary B., married, aged 50, was admitted into the Convalescent Hospital at Cheadle on April 2nd, having been transferred thither from the Manchester Infirmary, where she had been under the care of Dr. Simpson—suffering from Bright's disease and mitral stenosis. During her stay at the Infirmary she had some œdema and ascites, which, however, had entirely disappeared on her admission into the Convalescent Hospital. On the morning of April 16th she had an apoplectic attack, ushered in by loss of consciousness; and when this had passed away, it was found that the patient had some amount of aphasia, which was, however, but transient, and left hemiplegia. Two days after the attack I saw the patient, in conjunction with Dr. Grant, the resident Medical Officer at Cheadle, and noticed the following conditions. The patient has left hemiplegia, complete as far as the arm and leg are concerned, the face is but little affected. She complains of numbness in her left side and the sensibility seems slightly diminished on that side; her intelligence is clear, her speech but little affected. Occasionally she cannot find the exact word she wishes to express, but otherwise speech is unaltered. She complains of not being able to see well, and on examination it is found that she has left hemianopsia, the hemianopsia in each eye not reaching quite up to the point of fixation, but coming very near to it. The line limiting the hemianopsia is almost vertical, and the loss of vision within the area of hemianopsia absolute. The central vision of the patient is fairly normal, the fundus of the eye normal, the pupils are equal and react well to the light. There is no affection of any other of the cerebral nerves. The other special senses are normal.

The tendon reflexes on the paralysed side are slightly increased. There is no paralysis of either bladder or rectum. The area of cardiac dulness is increased, and over the apex a distinct systolic blowing murmur is heard, very limited in

extent; the second aortic sound is much accentuated. The urinary secretion is profuse, and contains albumen and granular casts. The temperature of the body since the attack has been above the normal, showing evening exacerbations.

I saw the patient again on the 23rd of April, and found her in almost the same condition as on the previous visit, except for the hemiplegia, which was now less marked, the patient being able to move her hand and forearm slightly. The hemianopsia had remained in the same state. The patient complained of neither headache nor giddiness.

She remained in this state till April 29, when she became somewhat suddenly drowsy, then comatose, and died the same day.

The post mortem was made on April 30 by Mr. Kaye, resident Clinical Clerk at Cheadle, who found the kidneys small and granular; the heart very much hypertrophied, especially the left ventricle; the mitral valve narrowed, but without any endocarditic deposits. The brain and its membrane showed externally normal relations, except the left lobe

Fig. 2.

of the cerebellum, where a freshhæmorrhage was found on the under surface of the lobe, just beneath the pia mater, forming a thin layer of about two lines in thickness and an inch in circumference. On making successive sections through the brain, the following lesions were found: Two small spots of hæmorrhage, *h. h*, Fig. 2, circular in outline and of ½ inch diameter in the

centr. ovale of right hemisphere, corresponding to the ascending frontal convolution. (The accompanying drawing, Fig. 2, made by Mr. A. H. Young, shows these lesions in M. Pitre's diagram.) A little behind these two hæmorrhages, corresponding nearly to the ascending parietal convolution, was another small hæmorrhage, smaller than the two just described, but again situated entirely in the centr. ovale, encroaching neither upon the cortex nor upon the basal ganglia. On making further sections through the brain, a small circular hæmorrhage, measuring about four lines in the larger transverse diameter and three lines in the vertical direction, was seen at the posterior and upper extremity of the right thalamus opticus, the part described as the pulvinar, not, however, reaching the upper surface of the thalamus, and therefore quite within its substance. The corpora quadrigemina, the corp. genicul., and the optic tract are found perfectly intact, nor did the rest of thalamus in the immediate neighbourhood of the hæmorrhage show any changes.

The diagnosis made during life was: "hæmorrhage into the right thalamus opticus, affecting the internal capsule," and it was thought that there was only one hæmorrhagic focus.

The post-mortem examination showed that there were several spots of hæmorrhage, that the internal capsule was perfectly intact, and that the hemianæsthesia and the hemiplegia were due to separate hæmorrhages; for on comparing the symptoms during life with the lesions observed after death, I think there can be little doubt that the following is the correct view. The hemiplegia was due to the several hæmorrhages in the centrum ovale, for these are situated in that part of the white substance which contains the motor fibres radiating from the internal capsule to the motor area of the cortex (see Fig. 2). The hemianopsia was due to the hæmorrhage into the thalam. opticus. The hæmorrhage on the under surface of the left lobe of cerebellum, which was of more recent date than the other hæmorrhages, cannot well be connected with either the hemiplegia or the hemianopsia; it probably came on the last day of life, when the patient was noticed to become drowsy and comatose.

What makes this case especially interesting is the definite and well-circumscribed lesion in the thal. optic. with which the hemianopsia must be connected. *Gowers* has observed hemianopsia in many cases of cerebral hæmorrhage independently of the seat of lesion; in all these cases, however, the hemianopsia was temporary only, and soon passed away. In our case, on the other hand, the hemianopsia was permanent and, as regards its extent, constant.

Now it is admitted on all hands that fibres from the optic

tract (chiefly of its external root) pass either directly or indirectly (through the external geniculate bodies, and according to some also through the anterior corp. quadrigem.) into the thalam. opticus, and thence to that portion of the cortex of the brain which forms the visual centre. Some other fibres, however, particularly those of the internal root, and the fibres described by Stilling as passing into a ganglion situated in the peduncle of the brain, do not traverse the thal. optic. If one may therefore draw conclusions from this case, it would seem that nearly all those fibres of the optic tract which are concerned in vision pass, after having undergone semidecussation in the chiasma, whatever their connection with the corp. quadrig. and corp. genicul. may be, almost in their entirety through the thal. optic. on their way to the cortical visual centre.

Looking over the literature of the subject, I find that there are a few cases on record in which lesions of the thalamus opticus were associated with hemianopsia; in most, if not all, the lesions were more extensive, and implicated neighbouring parts.

In Pooley's case,[1] where there was right paresis, right hemianæsthesia and right hemianopsia, a tumour was found in the occipital lobe reaching to the cortex; the thalamus opticus was found softened.

Baumgarten[2] describes a case of left hemianopsia coming on suddenly in a patient affected with Bright's disease. Death ensued after several months. An old apoplectic cyst was found in the right occipital lobe and thalam. opticus.

The only other point I wish to draw attention to in our case is the condition of the pupil. This was found to react well to light, which would be quite in accordance with the view held by many that the centre for the reflex movements of the iris is situated neither in the cortex nor in the thalam. opticus, but most probably in the anterior corp. quadrigemina. I mention this, as it is important from a diagnostic point of view in distinguishing hemianopsia produced by a cortex lesion from hemianopsia produced by lesions of the optic tract, or of the ganglia situated in front of this reflex centre.

[1] Knapp's 'Arch.,' Bd. vi. p. 27. [2] Centralb. f. med. Wissen.

NEUROMA OF THE MEDIAN NERVE REMOVED BY OPERATION.

BY WALTER RIVINGTON, F.R.C.S. ETC.

THOMAS B——, 29, labourer, was admitted into the London Hospital on December 22nd, 1873, with a tumour of the right arm. It occupied the inner aspect of the arm a little above the centre, and was about the size of a cricket-ball. It was hard, resisting, and only slightly elastic. It was very tender, and caused the most severe pain, which radiated along the limb, affecting especially the dorsal aspect of the middle and index fingers. No pain was felt in the palm. On the 28th of December the patient was placed under the influence of chloroform, and Esmarch's bandage was applied. A longitudinal incision was made over the tumour, through the skin and fasciæ, an investing capsule was opened, and I was then able to separate the tumour from its attachments, and to turn it out of the capsule without interfering with the nerve with which it was connected. It was bilobate, being crossed in the centre by a transverse constriction, and it had tapering prolongations at each end which seemed to attach it to the median nerve below, and to one of the cords of the brachial plexus above. Some of the loose outer parts of the capsule were removed; but I preferred leaving the greater portion of it to running any risk of interfering with the important nerves in relation with it, by endeavouring to dissect it away. A portion of the sheath subsequently sloughed, and came away. An attack of erysipelas of the *face* followed the operation, but it did not last more than a few days, and the wound healed kindly enough. At the beginning of February the patient complained of weakness of the muscles of the forearm, and galvanism was applied with benefit.

No cause could be assigned for the growth of the tumour. Two years previously the patient had fallen down a ship's hold, but he could not say whether he struck the arm or not. He first noticed the swellings six months afterwards, when it was as large as an egg. It began to be painful five months before his admission.

On cutting the tumour across, it was found to be of firm texture, and generally of a greyish-white colour, with some small yellow patches near the margin. These patches were thought to be portions of the growth undergoing fatty degeneration, but on microscopic examination they were seen to consist of small spindle-cells, mingled with fibrous tissue. The general mass of the tumour appeared to consist of fibrous tissue, and a smaller proportion of spindle-cells than was found in the aforesaid yellow patches. The tumour was more or less vascular, especially in the yellow portions. It would therefore be proper to describe the growth as intermediate between a fibroma and a spindle-celled sarcoma, or as a fibro-sarcoma.

The patient was seen about two years after the operation. He had been sent into the hospital from the out-patients, and had been admitted under Mr. Maunder. He caught sight of me in the ward, and, beckoning to me, introduced himself, and showed his arm. There had been no recurrence of the growth, and he had good use of his arm, which he said was free from pain and had gained in strength. He had come in for a small tumour at the upper and inner part of his right thigh.

Remarks.—The only remark which I wish to make on the case is to particularise the great facility afforded to the surgeon by Esmarch's bandage in the removal of tumours requiring careful and delicate dissection. Vessels and nerves can be exposed and traced, if necessary, with as much accuracy as in the dissecting-room, and the exact attachments and connections of a tumour can be made out with a precision which is impossible when blood is effused and smeared over the exposed structures.

Abstracts of British and Foreign Journals.

Semon on Abductor Laryngeal Paralysis.—Dr. Felix Semon (*Archives of Laryngology*, No. 3, July, 1881) discusses the fact, which has been noted by various other observers besides himself, that the abductor muscles of the larynx have a special proclivity to paralysis, whether in central or peripheral disease of the vagus, spinal accessory, or recurrent laryngeal nerves.

With the exception of the crico-thyroid, as is well known, all the laryngeal muscles, adductors as well as abductors, are supplied by the recurrent laryngeal, which gives off the individual muscular branches when close to the larynx. But that there is differentiation of the centres and fibres for the various muscles is shown by the fact—and 22 cases are quoted—that in slowly progressive lesions, whether of the centres, or peripheral nerves supplying the larynx, the posterior crico-arytenoid muscles may be paralysed alone, or at least before the others are appreciably affected. On the other hand, he has not been able to find any case of *organic* disease of the nerve-centres or trunks, in which the abductors only were paralysed, though in functional neuroses the adductors are more particularly affected.

As to the cause of the special selection of the abductors in organic disease, if of central origin, we might explain it by a differentiation of the abductor from the adductor centres, though the question would remain, why should the abductor centres be specially affected? Various hypotheses might be entertained as to this, viz. the mutual co-operation of the adductor centres giving them greater resisting power than the isolated abductors; the abductors being more automatic and less under volitional control; and lastly, the analogous fact, that in central diseases the extensors are sooner affected than the flexors.

But as the same phenomenon occurs also in peripheral disease of the nerve-trunks, we may suppose either that the disposition of the nerve fibres is concentric, and the abductor fibres possibly situated at the periphery; or that there is a specific vulnerability

of the abductor filaments; or that possibly the adductors receive an increment of nerve force from the superior laryngeal nerve.

But all these hypotheses, he argues, are more or less open to objection, and he does not think that at present a satisfactory explanation is possible. Practically, however, the facts are of great value. If the vocal cords are immovable in the phonatory position, all the possibilities should be considered which might cause affection of the entire laryngeal nerve; immobility of the cords in the position of deep inspiration, if not occasioned by mechanical impairment or myopathic affection, is probably due to a functional neurosis, or local affection of the adductor twigs.

Goltz on the Functions of the Brain. Goltz (*Verrichtungen des Grosshirns; Pflüger's Archiv für Physiologie*, Bd. xxvi. Heft 1 and 2) publishes a further series of experiments on the cerebral hemispheres of the dog, in which, instead of following his old method of destroying the cortex with a stream of water, he has established the lesions by means of a White's boring machine, the construction of which is described. Several chromolithographic plates are given of the lesions and the symptoms which were observed in the different animals experimented on.

The general conclusions he arrives at are:

1. The hypothesis of circumscribed centres for special functions in the cerebral cortex is untenable.

2. There is no area of the cortex exclusively concerned with sight, hearing, smell, taste, or touch.

3. It is impossible by any circumscribed lesion of the cortex to produce permanent paralysis of any muscle, or remove it from the influence of the will.

4. The vital manifestations which we regard as indicative of intelligence, feeling, emotion, instinct, are not dependent on functionally differentiated cortical regions.

5. Destructive lesions of the frontal regions of the hemispheres cause defects which differ in certain respects from those caused by lesions of the occipital regions. These differences depend probably on simultaneous lesion of the conducting tracts which lead to the crura.

Seguin on Cerebral Localisation.—Dr. E. C. Seguin has reported several cases of considerable interest and importance as to the effects of localised cerebral lesions. His cases and remarks are contained (1) in a paper contributed to the *Transactions of the*

American Neurological Association, vol. ii., 1877, and (2) in another paper published in the *Journal of Nervous and Mental Disease*, vol. viii., July 1881.

He gives the particulars, and describes and figures the lesions in twelve cases in all. For these, reference must be made to the original papers.

The conclusions which Dr. Seguin has arrived at from these cases are :—

1. That the motor area of the cortex and allied white substance extends anteriorly as far as the lower half of the second and first frontal gyri, and posteriorly as far as the anterior part of the intra-parietal fissure. [Three cases referred to.]

2. The region lying between the limits indicated above, the middle regions of the hemisphere, on its convexity and (to a certain extent) on its median surface, including the posterior parts of the first and second, the whole of the third, frontal gyri, the whole of the ascending frontal and ascending parietal gyri, with their terminations in the longitudinal fissure known as the paracentral lobule, with probably the upper parietal lobe,—all these cortical parts, with their associated segments or fasciculi of white matter, have strong motor functions, being in direct relation with the muscles of the face, tongue, arm and leg.

This general statement is supported by the remaining nine cases, in which destructive lesions of this area gave rise to spasm or paralysis on the opposite side of the body.

He concludes more particularly :—

a. That the lower part of the third frontal gyrus is intimately connected with the organs of speech (and the function of language). Four cases are appealed to in support of this proposition.

b. The middle parts of the ascending frontal and ascending parietal gyri are directly connected with the arm of the opposite side. Two cases are cited in illustration.

c. The upper or posterior part of the ascending frontal and ascending parietal gyri, and the paracentral lobule (also the upper parietal lobule?) are directly connected with the lower and upper extremities of the opposite side, and perhaps more closely with the leg. Three cases are referred to in support of this proposition.

Tripier on the Cephalic Souffle in the Adult.—Tripier (*Revue de Médecine*—three communications—February, March, and October 1881) describes a cephalic souffle, said generally to be only

audible in children, which he has observed in the adult in cases of anæmia, chlorosis, and also in a case of intracranial tumour and in one of hydrocephalus. It is a deep systolic sound, which is diffused over the whole cranium, but which is heard in greatest intensity in the orbital region—by placing the stethoscope on the globe of the closed eye. In his first two communications he regarded the right temporal region as the point of maximum intensity, but in his last communication he states that it is the orbital region where it is best heard.

The sound is modified by pressure on the carotid, and this is perceptible both to the patient, who is aware of a subjective sound in the ear synchronous with the bruit, and to the observer. The souffle diminishes or disappears with the cure or aggravation of the disease.

There is no relation between the cephalic and cardiac souffle heard in such cases, nor is it originated in the arteries or veins in the neck.

He is of opinion that it originates in the internal carotid at the point where it enters the cranial cavity. In a case where there was a small tumour pressing against the artery here, a cephalic souffle was heard, and there was no cardiac souffle as in anæmia and chlorosis. From communication between the carotid and cavernous sinus, aneurism of the carotid or ophthalmic artery, the cephalic souffle can be diagnosed by the absence of, characteristic ophthalmoscopic appearances in the fundus, protrusion of the globe, and other signs.

The cephalic souffle does not exist in healthy adults, and Tripier believes that it may prove of great value in the diagnosis, prognosis and treatment of the cases in which it occurs.

Baginsky on the Semicircular Canals.—Baginsky (*Du Bois-Reymond's Archiv für Physiologie*, 1881, Heft 3 and 4) describes a series of experiments on rabbits and pigeons with a view to determine the nature and causes of the disorders of equilibrium and other phenomena which have been found by Flourens and other observers to be associated with injuries of the labyrinth. He injected liquids of various kinds into the tympanic cavity of the rabbit, and cut the semicircular canals individually on one or both sides of the pigeon. The results in both animals were similar. Injections of liquid always caused nystagmus and distortion of the head, temporary, or lasting till death, according to the intensity of the irritation. He is of opinion that the phenomena are due to irritation of the brain or to actual anatomical lesion. In pigeons

in most cases there were pendulum-like movements of the head, twisting of the neck, and frequently nystagmus, coming on directly after the injury of the labyrinth, or showing themselves a few days after. The cause of these he regards to be extravasations in the brain, or inflammation excited by the lesion. The pendulum-movements of the head in pigeons, which are not observed in rabbits, he attributes to peculiarities in organisation, and in reality are of the same nature as the distortion of the head seen in the latter. As to the exact way in which the brain is affected, he holds that by injections into the tympanic cavity the pressure in the cranium is suddenly increased, and this suddenness irritates the medulla oblongata more particularly at the point where the restiform body is in closest relation with the ascending root of the trigeminus. If the pressure is very great, sudden death may be caused by the effect on the medulla oblongata. Section of the semicircular canals in pigeons diminishes the intracranial pressure. The suddenness of this acts as an irritant on the brain.

The path by which the influences are conveyed from the labyrinth to the brain differs in the rabbit and pigeon. In the former it is by the perilymphatic apparatus and aquæductus cochleæ; in the latter it is by the endolymphatic apparatus and aquæductus vestibuli.

Baginsky is of opinion that the hypothesis of any special relation of the semicircular canals to equilibration or the sense of space must be given up, as all the phenomena connected with their lesion are to be attributed to injury of the brain.

Spamer on the Semicircular Canals.—Spamer (*Pflüger's Archiv*, Bd. xxv. Heft 3 and 4) criticises Baginsky's hypothesis, and points out among other things that the disturbances of equilibrium vary with the canal injured. There is no relation between the amount of disturbance and the amount of cerebrospinal fluid which escapes when a canal is cut. The disturbances vary with the kind of injury inflicted, being more marked on transverse than longitudinal section. Apart altogether from section of the canals, irritation by different chemical solutions causes marked results; and the effect of touching the canals with a heated point cannot be explained away by any hypothesis of direct injury to the brain, such as Baginsky assumes. In his own experiments the most careful examination failed to detect any lesion of the brain, or if such lesions were accidentally produced, the cases were considered unsuccessful experiments.

Spitzka on the Cerebellum.—Spitzka (*The Functional and Morphological Relations of the Cerebellum.* Reprint from *Chicago Med. Review*, July 1881) endeavours to arrive at the functions of the cerebellum by a consideration of its relative development in different animals, and of its structural relations. The cerebellum is not, as has been frequently stated, more highly developed in the lower than in the higher animals. The contrary is the case. The cerebellar tracts, viz. anterior, middle, posterior peduncles, auditory nerve-roots and trapezium, are all centripetal. The first two, though efferent as regards the cerebellum, are afferent to the cerebrum. Hence it would seem that a direct motor innervation is entirely excluded from the functional possibilities of this organ. The restiform tract is derived from the posterior columns of the spinal cord and direct cerebellar tract, and passes to the cortex of the cerebellum, a small portion, according to Stilling, going to the nucleus dentatus. On the inner side of the restiform tract there is a bundle of fibres, which he regards as being the ideal continuation of the columns of Burdach. These go to the nucleus fastigii of the vermis. The auditory nerve-root sends fibres both to the nucleus dentatus and n. fastigii. These fibres are interrupted by the auditory nucleus, and the connections between the cerebellar ganglia and the auditory nucleus are more numerous than the fibres of the auditory nerves themselves, indicating an important connection between the internal ear and cerebellum.

The trapezium joins the restiform tract. It appears to be derived from the tegmental fibres of the medulla oblongata, and goes to the vermis and nucleus fastigii. Perhaps also the fifth and pneumogastric nerves have direct cerebellar connections. The anterior peduncles (præpedunculi) run from the nuclei dentati and decussate with each other into the nuclei tegmenti. The middle peduncles (pontibrachium) form the transverse fibres of the pons, and are continued to the cerebrum in the pes pedunculi. Besides these there is an important connection, not yet clearly made out, between the cerebellum and corpora quadrigemina, viz. the valve of Vieussens, which probably has a special relation to visual co-ordination.

From a consideration of these structural connections, Spitza is of opinion that the cerebellum, whether the lowly vermis of reptiles and marsupials, or the highly developed organ of the higher animals, constitutes a sort of deflection or meeting-point for impressions originating in the auditory and tactile end-organs. Its main object must therefore be the fusion of these sensations, or

their correlation in some way for the benefit of the cerebral hemispheres. The cerebellum is not to be considered the centre for, but "the informing depôt of the finer co-ordinations engaged in maintaining the equilibrium." The cerebellum cannot act per se, but only in connection with the higher centres.

If it is asked why the sensory tracts going to the cerebellum should not go direct to the cortex, the answer is that the position of the associating ganglion of the auditory and tactile impressions was primarily conditioned by the termination of the auditory nerve-root. "Just as the cerebellum may be considered at least in its higher form as a luxuriant hypertrophy superimposed on the auditory ganglion, so the cerebrum is known to be a supplantation of the olfactory lobe."

Féré and Demars on Ménière's Disease and its Treatment.—Féré and Demars (*Revue de Médecine*, Oct. 1881) describe the symptoms, and give the details of several cases of Ménière's disease with particular reference to the influence of quinine on them, according to the method suggested by Charcot.

When a patient with Ménière's disease is treated with sulphate of quinine in doses of from 60–80 centigrammes (10–12 grains) the cinchonic sounds in the ears are added to the subjective sounds of the disease, and the patient's symptoms are rather increased. But if the quinine is omitted for eight or ten days, the sounds and the vertigo diminish notably. If the quinine is readministered for a similar period of from eight to ten days, there is a new exasperation of the symptoms, but of less intensity than at first. At the second cessation there is a very considerable amelioration, and this may go on to complete cessation of the vertiginous attacks.

Kolnessikow on the Pathology of Hydrophobia.—Kolnessikow (*Virchow's Archiv*, Bd. lviii., Heft 3, Sep. 1881) discusses the morbid appearances found in hydrophobia, and after a resumé of the results of other observers, proceeds to give an account of the changes he has observed in 20 dogs examined by him in the three years 1878–1880. His investigations relate exclusively to the condition of the brain and nervous system—the condition of the other organs and viscera being entirely in accordance with the observations of Rudnew, Lange and others.

Macroscopically the condition observable most commonly was a hyperæmia of the pia mater of the brain and cord. In a few cases there was an œdematous condition.

The microscopical appearances of the individual cases he generalises as follows:—

The changes are found regularly and chiefly in the walls of the blood-vessels. The vessels are distended with a mass of red blood-corpuscles, and the vascular walls, as in all acute encephalitic processes, exhibit proliferation of the nuclei and infiltration of the whole thickness of wall with round cells. The round-cell infiltration was seen also along the vessels and round the nerve cells in the cerebral hemispheres, spinal cord, and medulla oblongata, in insular patches. These changes in the vascular walls and interstitial tissue vary in intensity, probably dependent on the intensity of the infection, and the relative duration of the malady. The changes are particularly manifest in the corpora striata, medulla oblongata, and spinal cord. They are not so pronounced in the hemispheres, and are more frequently circumscribed than diffuse. In all cases there were found between the layers of the vascular wall and around them a large number of amyloid or hyaloid, occasionally pigmented, colloid exudation flakes and conglomerations. These consisted partly of extravasated fluid elements and blood-corpuscles, and partly of infiltrated lymphoid elements which had traversed the vascular wall. With the change in form there was also a chemical change, as most of these elements became amyloid in character, and a few colloid and pigmented. Besides these flakes and masses, there were more rarely in certain vessels, protoplasma elements (the cells of the adventitia), which contained in their protoplasm red blood-corpuscles and finely-granular pigment.

Meyer on Diphtheritic Paralysis.—Paul Meyer (*Virchow's Archiv*, Bd. lviii. Heft 2) describes the appearances he has met with in a well-marked case of diphtheritic paralysis. He has found that the peripherical nervous system exhibits well-marked signs of parenchymatous neuritis, the characters of which he describes in detail and figures.

The alteration was best seen in the branches to the diaphragm and to the abdominal muscles (including the cremaster). Next to these came the nerves to the ilio-psoas, deltoid, triceps; then those of the glutæi and adductors of the thigh. After these came the nerves to the quadriceps, and the muscles of the forearm and leg. In general the alteration is very diffuse, and there is no special predilection for any particular group, such as flexors or extensors.

Degeneration was also found in the cutaneous nerves, but to a less extent than in the motor branches.

The cranial nerves also exhibited well-marked signs of degeneration, specially well seen in the oculo-motor, abducens, and glosso-pharyngeal.

In the large nerve-trunks the degeneration was not so pronounced as in the nerves of the face and neck. The degeneration appeared to be inversely proportional to the calibre of the nerve. Degeneration was also visible in the anterior and posterior spinal roots, more particularly in the cervical and lumbar region than in the dorsal. The anterior roots were relatively more affected than the posterior.

Degeneration was also to some extent evident in the multipolar cells of the cervical and lumbar region of the cord. The cells of the posterior cornua were also affected to some extent.

The author concludes that the diphtheritic infection attacks the nervous system in general, both spinal and peripheral, and that there is no reason to regard the one as secondary to the other.

DAVID FERRIER.

Leegard on the Degeneration of Nerves and their Electrical Reactions.—(*Arch. Klin. Med.* 1880.)—It is notorious that writers differ greatly in their accounts of nerve degeneration and regeneration. This may partly depend upon the nature of the primitive lesion. Our author employed only the ligature, which he carefully applied to the nerve without disturbing its natural relations. The thread is tied, and immediately removed, so as simply to produce a solution of continuity in the axis and myelin, the empty sheaths remaining intact. The microscopical examination of the nerves was made on specimens macerated for twenty-four hours in osmic acid, and then stained in picrocarmine and other agents. In three or four days the myelin begins to run together into cylindrical masses. The axis breaks at places; hence the loss of electrical excitability. *Secondary* to these changes appears an enlargement of the nuclei in the sheath. The myelin gradually undergoes further segmentation, and ultimately (three weeks) granular degeneration. The axis breaks up also, and every trace of it is lost between the tenth and fifteenth day.

There is an increase in the number, as well as in the size, of the nuclei. At both extremities they present masses of protoplasm which tend to form a continuous streak along the sheath. This is the first step towards regeneration. Eichhorst and Neumann thought that the products of disintegration of the medulla and axis underwent a kind of chemical alteration, and that this new sub-

stance persisted in the sheath. But it is more probable that those products are altogether absorbed. It is very likely that some fibres are completely destroyed, sheath and all, and that this is the origin of the granular débris noticed among degenerating fibres.

The process of degeneration does not follow a centrifugal direction, but attacks the whole peripheral portion of the injured nerve at once. At the point of injury itself there may be some inflammatory changes, if the operation has not been conducted with due care; otherwise there occurs nothing but an increase of the nuclei. Immediately above this point the central end of the divided medulla and axis offer much the same alterations as the peripheral end on a length of 3–5 mm. or more. The first interannular segment is altered as above described; the next three or four are shorter, irregular, with their medulla broken up. The sheath is empty at places, and displays nuclear proliferation.

As mentioned above, the first steps of the process of regeneration are made before the degenerative process is complete, and starts in the protoplasma accumulated within the sheaths in the neighbourhood of the nuclei. Fine greyish threads become differentiated in the interannular segments, and run together into a continuous fibre. The new segments appear to be more numerous than in the healthy nerve. It is possible that wholly new fibres may be reproduced, but this is not certain. The supposition that several new fibres may originate in a single original one is baseless. The regeneration is the more rapid, the more peripheral the lesion has been.

The irritability of the *nerve* undergoes the following changes. There is frequently a slight increase of it during the first day after the operation. This, however, is not constant, and is followed by a diminution which becomes more marked until the nerve does no longer react to the most powerful stimuli. This occurs, on an average, on the third day. The period of unexcitability lasts a variable time, according to the depth of the lesion; and voluntary motion, as we know, returns before any electrical reactions can be obtained, especially below the point of lesion. Applied here, the galvanic current sometimes brings about muscular contractions before the faradic. It is to be noticed, also, that opening contractions are more difficult to obtain than in the normal state. The author confirms the few observations previously recorded, where an inversion of the polar effects on the nerve is noted; he has found that in certain cases A.C.C. $>$ K.C.C., and K.O.C. $>$ A.O.C. In these, he says, every experimental error has been carefully

eliminated, but he cannot give any explanation of the occurrence. The indirect stimulation was always characterised by a rapid, healthy contraction.

With reference to *muscle*, its loss of reaction to faradism is usually complete within four days. Vulpian has rightly insisted that this is not strictly true of muscles directly excited (not percutaneously), for here fibrillary contractions persist for a long time. Section of the antagonists may be necessary to demonstrate weak contractility. To galvanism muscles at first react less freely, soon after the injury to the nerve, but this applies less to the strength of the current to be used than to the amplitude of the contractions; in other words, it is the maximal, not the minimal, test which displays the alteration. At the same time A.C.C. approximates K.C.C. The author has not seen the same regularity in the development of the qualitative changes during the period of increased excitability, as is usually described; he has never seen A.C.C. > K.C.C. Both O.C. disappear at first; but when they return A.O.C. is always > K.O.C. They tend to overtake the C.C. during the period of increased excitability, and disappear again afterwards, sometimes permanently. During the same period the closure and opening tetanus appears only after the closure and opening contraction have been obtained. A singular fact is that the author has not been able to obtain signs of the increased mechanical excitability of muscles so generally found in the human subject, nor of Erb's "middle form" of the degenerative reactions. The course of the electrical phenomena connected with the processes of nerve and muscle degeneration may be divided into four stages, of which a tabular view (p. 565) is appended.

[The reader interested in the question will find an account of the similar process as observed in man in the reporter's "Introduction to Medical Electricity," Chapter III.; or better still in Erb's vol. on the Peripheral nerves, in Ziemssen's 'Cyclopedia.']

Langenbuch on Nerve Stretching in Ataxy and other Spinal Diseases. (*Berlin Klin. Woch.* 1881, 24–27.) — The author begins by expressing the view that locomotor ataxy is not primarily an affection of the posterior tracts, but has a peripheral origin in the sensory nerves. In all the cases in which he operated, the nerves showed, to the naked eye, signs of alteration; sometimes swelling and reddish discoloration, sometimes dryness, atrophy, and yellowish or violet discoloration. He thinks that the large number of cases of tabes, as well as the great importance

—	First Stage.	Second Stage.	Third Stage.	Fourth Stage.
Nerve.				
Motility	Absent	Absent	Returning	Good.
Reaction to galv. and far.	Increase at first, then disappears.	Absent	Returning	Further improvement. Normal formula restored.
Muscle.				
Farado-contractility	Disappears; only fibrillary contraction.	Fibrillary only	Reappears at some period	Rises, but not to normal.
Galvano-contractility	Diminution of C.C. Disappearance or inconstancy of O.C. Tetanus diminishes.	Increase of all C. to maximum of hyperexcitability. O.C. constant. Tetanus evident, but appears last of formula.	At first increase, then decrease of C.C. below normal. O.C. decrease and disappear. Tetanus diminishes.	Diminished irritability. C.C. recover. O.C. absent or inconstant. Eventually rise of irritability and return of normal formula. Irritability may remain permanently deficient.
Character of contracting	Rapid. Maximal often very weak.	Slow, vigorous	Strength falls off. To indirect stim. rapid, to direct slow. Gradual improvement of latter.	Strength restored; sluggishness disappears.
Mechanical irritability	Diminished	Exalted (?)	Exalted (?); later diminished	Usually diminished.

of cold in the causation of disease, is rather in favour of the peripheral origin. Another consideration is the fact that in the early stage the morbid changes in the cord are limited to the part of the posterior tracts which Flechsig has shown to be the direct continuation of the peripheral nerves. The pathological process invades Goll's columns at a later period: these we know are not in direct connection with the peripheral fibres. Further extension occurs later, by contiguity, into the neighbouring lateral tracts.

According to Langenbuch, then, we have to do in ataxy with an ascending inflammatory alteration in the nutrition of nerves, roots, and columns. How is it then that the overgrowth of connective tissue (sclerosis) is proportionally much larger in the cord than in the nerves? He thinks that the answer to this question lies in the fact that the morphological character of the connective tissue is different in the two cases. The result of connective overgrowth is much more important in the cord where the nerve elements are thickly crowded together among the thin meshes of the tissue. The author is, besides, far from saying that the morbid process always invades the cord. This is well illustrated in the temporary ataxies after acute disorders. With reference to the lightning pains, their peripheral origin seems to be shown by the happy results of compression or friction along the course of nerves.

The action of nerve-stretching is twofold: it stimulates the conductivity of partially disabled nerve-fibres, and promotes resorption of morbid products. It seems to influence the whole nervous system; the pulse and respiration-rate is quickened.

The first symptom to give way is the sensation of chilliness. After a few hours, sensibility returns in the limbs, and may even reach hyperæsthesia. After a certain diminution, muscular power increases, and the ataxic symptoms disappear. During the first few days peripheral pain is present, perhaps as the result of the operation; but within the second week the patient usually becomes entirely free from pain. The symptoms connected with the rectum and genito-urinary organs are also usually much relieved. A curious phenomenon sometimes observed is profuse perspiration after the operation. The general vigour, physical and mental, of the patient is increased, but the gait remains abnormal for a certain time. The knee-phenomenon remained absent in the twenty-two cases in which the author operated.

He has performed nerve-stretching in various other cases of spinal disease—in one (lateral sclerosis) the result was very favour-

able. Two cases of general chronic pemphigus and obstinate senile pruritus were cured by the stretching of one sciatic nerve.

On the retardation of Sensory Impulses in Locomotor Ataxy. FISCHER (*Berlin Klin. Woch.* 1881, 33) examined five cases of tabes with a view of determining the relative rate of sensations of touch and pain. For such experiments it is advantageous to deal with patients intelligent enough to give an accurate description of their feelings. The retardation for impressions of pain may be considerable. He found it once to extend to fifteen seconds, but states that sensations of touch may be delayed longer than those of pain. The delay may vary during the experiment; it may become less on repeating the stimulus; or, contrariwise, the stimulus may cease to be perceived at all. Retardation of the other kinds of sensations (temperature, pressure, &c.) was not clearly demonstrated. The author does not offer any theory in explanation of the retardation.

Unilateral Perspiration and Hemiatrophy, with their relation to the Nervous System. TAKÁCS (*Ctrlblatt. f. Nervhlkd.* 1881, 13) has observed a case in which the patient (a man, aged 32, left-handed) was seized with pains, weakness and stiffness in the right extremities. At the same time he ceased to perspire on the right side of the body. Pilocarpin injections produced some sweating on that side, especially on the back of the hand, where perspiration appeared more abundant than on the left hand. The whole right side of the body was, when patient came under observation, viz. two years after the beginning of the disease, much thinner than the left. There was distinct atrophy of all the right hand-muscles, which showed deficient reaction to electricity.

The author remarks that most cases of a similar nature hitherto described have been referred to hyper- or hypokinetic conditions of the sympathetic. He thinks, however, that the combination of pupillary vasomotor and trophic symptoms has its origin in the cord itself. The cilio-spinal centre accounts for the pupillary symptoms. And, according to the late researches of Adamkiewicz, perspiration is governed by the anterior grey matter of the cord. The suspension of perspiration does not depend upon mere vaso-construction, but upon paresis of special nerve action, and pilocarpin is a stimulant of such an action.

Cherchevsky on the Laryngeal Crises of Locomotor Ataxia (*Revue de Médecine*, July 1881). The author describes eighteen

cases of tabes, in which laryngeal crises occurred. He classifies the latter in three groups, according to the intensity of the symptoms:

1. In the milder form there occur fits of coughing very like those of whooping-cough, consisting in a series of rapid short expiratory efforts, followed by a deep whistling inspiration. The duration varies from a few to ninety seconds. The expectoration is very scanty.

2. In the second group the cough is much more violent, and the external manifestations (congestion of the face and eyes) much more marked. The crowing inspiration louder and more difficult. Other symptoms, such as headache, nausea, vomiting, &c., accompany the fit. The duration is longer; five to ten minutes.

3. In the severest form there is complete apnœa, with symptoms of asphyxia, unconsciousness, epileptiform convulsions. There may be local prodromal symptoms before the fit. Its duration may extend to half an hour, or even several hours.

The laryngoscope reveals no local change; and the time and number of fits varies much in different cases. Post-mortem appearances in two cases pointed to atrophy of the vagus and accessorius roots, and of the posterior pyramids, besides the usual tabetic alterations in the cord.

In the majority of instances the laryngeal crises appeared along with the early symptoms of tabes; and occasionally formed the only prominent symptom for a considerable time.

A. DE WATTEVILLE.

A Microcephale of Simian character.—Professor Tamburini showed to a Medical Congress at Reggio Emilia a microcephalic boy, named Battista, now twelve years of age. He was a foundling. When three and a half years old he had been described by Professor Lombroso (see my report on some cases of microcephaly and cretinism in the *Edinburgh Medical Journal*, August, 1875, and my book on *Idiocy*, p. 87). Dr. Tamburini's paper is a reprint from the *Archivio Italiano per le Malatie Nervose*, Fasc. I., Anno 18, 1881.

Lombroso described Battista as having a tender and delicate skin, covered with fine hair on the forehead, limbs, cheek and neck. The head was small and peaked, with a retreating forehead, and the external orbital process protuberant. The face was prominent; the incisors distant from one another, and the canines twice as big as usual. The palate was longer, and the genital organs more developed than usual. The hallux more distant and longer than the other toes.

His height was 79 centimetres; the weight of the body 7 kilogrammes. The circumference of the head was 360 millimetres; the longitudinal curve 230 millimetres; the transverse 190; the longitudinal diameter was 128; the transverse 100. The cephalic index was 78°, and the facial angle 72°.

The most striking characteristic of this child was his fondness for leaping, and great restlessness. He generally leapt with the back bent and the hands in front of him. He did not sit, but crouched upon the ground. He smelt his food before carrying it to his mouth.

In 1877 he was brought to the asylum of Voghera, owing to his habit of tearing and destroying things and killing animals. Dr. Tamburini observes that his appearance and movements no longer answered to the picture given by Lombroso taken six years before. The smallness of the head and its disproportion to the body were very striking. The light hair diffused over the face had disappeared. His attitude and movements have still something of the Simian.

On making a number of measurements and comparing them with those of Lombroso six years before, the following differences were found on the body and head. The circumference of the head stood to the stature :—

In 1871, as 45 to 100;
1877, as 36 to 100;
1880, as 32 to 100.

The circumference of the head in 1877 was 385; in 1878, 395; in 1879, 400; in 1880 it was 418. The head was oxycephalic, and there was a marked want of symmetry in bulging behind on the right side and in front on the left. Battista was very restless, and his attention was difficult to fix; he knew the names of many things, and even used phrases; but his ideas went no further than the objects around him, and he could not be taught to read or write. He was fond of music, had little attachment to particular persons, and was subject to fits of furious passion. The genital organs were more developed than that of an ordinary boy of the same age, and his sexual desires seemed to correspond.

The Optic Tracts and the Visual Centres.—Tartuferi has in the Pathological Laboratory of Turin conducted a series of experimental researches on the optic tract and the visual centres in the cerebral ganglia and cortex cerebri. From a note in the *Italia*

Medica (No. 5, 1880) it appears that he has arrived at the following conclusions:—

1. Where the corpus geniculatum externum appears to be smaller it is often dependent upon the sclerosis of the optic tract which extends over it.

2. It is not true, as up to this time has been stated by anatomists, that all the fibres of the corpus geniculatum externum belong to the optic tract. It only forms a small part of its medullary cortex, and passes over it without entering into any intimate connection.

3. The corpus geniculatum externum cannot be considered as belonging to the mesocephalic centres of the apparatus of vision. Assuredly, at least, it has not the important significance which some anatomists would attribute to it.

4. The inferior commissure of Gudden is not, as its discoverer imagines, a simple commissure between the two optic thalami and the two corpora geniculata interna.

5. The greater part of the inferior commissure of Gudden, which is in connection with the two corpora geniculata, and especially with the external one, probably goes to compose the inferior portion of the superficial white and grey layer.

6. There are no fibres of the optic tract on the surface of the optic thalamus.

7. The thalamus is not a visual centre.

8. What is called the inner root of the optic tract going to the thalamus (innere Sehhügel-wurzel of Meynert, or tiefere Wurzelbundel of Studa) does not belong to the optic tract.

9. The fibres which compose the so-called root of the corpus geniculatum internum (Huguenin) does not belong to the tract, but to the inferior commissure.

It results from these four last conclusions that of the five roots of the optic nerve recently admitted (Huguenin, 1879) there is only one real root which passes over the corpus geniculatum externum without entering into it, and goes to the anterior tubercle of the corpora quadrigemina.

10. The diminution of the punctus occipitalis is due to atrophy, and not, as Gudden says, to a sinking, owing to the diminution of the anterior tubercles of the corpora quadrigemina which lie below it.

11. The point of the occipital lobe in the rabbit constitutes the cortical centre for the apparatus of vision. This has been confirmed by experiments upon monkeys, cats and dogs, as well as by pathological observations on man.

The effect of Metals on the Sphygmograph.—Professor Orsi, in the University of Pavia (*Italia Medica*, No. 8, 1880), studied two cases of hemianæsthesia which were modified and transferred by the application of different metals. He determined to try what effect the application of metals would have upon the sphygmograph, and came to the following conclusions.

a. That the application of lead to the skin during the first two minutes only sensibly lowers by a half, and in the end by a third, the ascending line.

b. That by substituting zinc for lead the line soon rises beyond the normal limit. If the application of zinc lasts for a while, there is a notable change in the height of the line occurring along with changes in sensibility and muscular force. The tracings obtained under the action of zinc are unequal in their character. In the ascending line they are of different heights and more or less sloping.

c. That the application of the magnet and electric current at a distance causes inequality and abnormality of the sphygmographic tracings, much in the same way as the hypodermic injection of from one to two centigrammes pilocarpin, which appears to have a stimulating action on the heart. WILLIAM W. IRELAND.

Rumpf on the Action of Lymph on the Nerve Centres. (*Pflüger's Arch.* xxvi., *p.* 415.)—Rumpf maintains the correctness of his observation that the axis-cylinder of nerve is dissolved by lymph, after passing through a preliminary stage of swelling. The swelling of nerve stumps under the influence of lymph is at first of the nerve proper, and only later consists in an inflammatory hypertrophy of the connective tissue. The author was led to examine the analogous event as regards fibres and cells of the brain and cord. The results on frogs are very striking:—the skull having been opened, a portion of brain isolated, and the wound reclosed, Rumpf found forty-eight hours later that nothing remained of the isolated portion of brain but a small quantity of connective tissue. The spinal cord having been laid bare, transected above and below, and isolated from all nerves, Rumpf found at the end of twenty-four hours that all the nerve elements have began to swell, this being still more marked at the end of forty-eight hours, after which the cord became amorphous and rich in nuclei, and (five to six days) disappeared, leaving nothing but a little connective tissue. This absorption does not ensue upon section of the cord without section of its nerves, nor, when the cord

and intervertebral nerves having been cut, the cauda equina is left intact. Nor is it produced if in addition all sensory roots of the cauda are divided; whereas if its motor roots are alone divided absorption follows, thought not so rapidly or completely. He obtained similar results with hardly any interference with the circulation. These observations indicate a centripetal trophic influence, partly via sensory channels, but chiefly via motor channels, and confirm the view of Kühne and of the author (1860) that from end-plate to centre there exists a constant excitatory and trophic influence.

The author also experimented on the brain of pigeons, and found, as in the case of frogs, swelling; absorption was, however, usually complicated by suppuration.

The author concludes that, to the maintenance of centres, their union with efferent channels is necessary, and that failing this union the organ degenerates and is absorbed by the action of the lymph; also that this union alone is sufficient to preserve the otherwise isolated cord, and that even partial union suffices by vicarious action to maintain the nutrition of the whole organ. This retrograde influence is the antithesis of tonus, and Rumpf therefore terms it "retrotonus." Thus, as laid down by Sigmund Mayer, the nerve fibre, with its two end-organs, central and peripheral, constitutes a trophic as well as functional unit.

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END OF VOL. IV.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET AND CHARING CROSS.